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JOURNAL OF NEUROSURGERY

[*J. Neurosurg.*]

VOLUME VII

1950

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INTRADURAL SPINAL GRANULOMAS

PAUL C. BUCY, M.D.,* AND H. R. OBERHILL, M.D.*
Chicago, Illinois

(Received for publication April 4, 1949)

INTRASPINAL granulomas are rare, but they do occur, and are most frequently the familiar extradural tuberculous granulomas associated with Pott's disease of the vertebrae. Occasionally there may be no associated vertebral involvement. Non-specific granulomas in the extradural location, though less common, have also been described.[23]

A granulomatous lesion of the dura mater itself, hypertrophic spinal pachymeningitis, was described in the 1870's by Charcot[1] and his pupil Joffroy. This condition is most common in the cervical region, but may occur elsewhere in the spinal dura mater. It is usually non-specific, but occasionally is said to be of tuberculous or luetic etiology.

Intradural granulomas are very rare and may be intramedullary or extramedullary. They may be of syphilitic origin in the form of a gumma, which now is a rare lesion anywhere in the body, or tuberculomas, which again are quite unusual lesions, or they may be of other etiologies. As a granuloma represents an intense reaction on the part of the tissues to a specific or non-specific infection or irritation, the varieties of granulomas possible are manifold. Among these are: granulomatous tumefactions of pyogenic, other bacterial and mycotic (blastomycotic, coccidioidal, torular, trichophytic) origin, as well as those caused by the irritation of non-infectious agents such as injected iodized oils, dusting powders, surgical cotton and other foreign bodies. All of these varieties, however, have not been found intraspinally. The eosinophilic granuloma, while reported as causing symptoms of the central nervous system,[11] is of secondary interest in this discussion in that symptoms produced would probably be those of extradural pressure secondary to the bony involvement.[8]

DISCUSSION

Tuberculoma of the spinal cord or its leptomeninx is a rare entity. In a series of 6000 postmortem examinations of the central nervous system at the Philadelphia General Hospital,[26] only 80 (1.3 per cent) cases of tuberculomas of the central nervous system over 1 cm. in diameter were found. Of these, 78 per cent were in negroes, 22 per cent in white people, 58 per cent in males and 42 per cent in females. There were 40 solitary lesions and 40 showed 2 or more granulomas present per case. In only 6 cases were there tuberculomas of the spinal cord, 3 solitary lesions and 3 associated with granulomas elsewhere in the central nervous system. Tuberculomas were found in the central nervous system in 1 out of every 53 cases that showed

* 25 East Washington Street, Chicago 2, Illinois.

evidence of tuberculosis elsewhere in the body. In 8 cases tuberculoma of the central nervous system was the only tuberculous focus anywhere in the body. The correct diagnosis of tuberculoma was made clinically in only 1 of the 80 cases.

The disease is seen in childhood or early adolescence rather than in later life in the majority of cases. The lesions vary in size from that of a small nodule a few mm. in diameter to that of a hen's egg, and produce the symptoms of any other space-occupying mass in the same location in the spinal cord. They are usually associated with foci of tuberculosis elsewhere in the body and are probably secondary to these.[24] These tumors may be intramedullary or extramedullary. The latter often arise from an infection of the spinal nerve roots, intervertebral cartilages, the epidural tissues, or the vertebral bodies. Peculiarly, the involvement of the spinal cord produces an inflammatory reaction of the pia arachnoid and dura mater, which may fuse all of these membranes into one thick mass and cause changes in the epidural space as well. These changes are most marked at the level of the tuberculoma and may include the adjacent nerve roots. The meningeal involvement may not spread far beyond the level of the principal lesion. It is very likely caused by the discharge of tuberculous debris into the subarachnoid space.[6] On gross inspection, a round mass with concentric layers is found. It may show a softened, necrotic center with some surrounding edema, but the center may be calcified or, on rare occasion, present the findings of a true abscess.

In unusual instances the lesion may be removed with good results, as was shown by Elsberg's[3] patient who lived more than 8 years after operation, but usually a spreading tuberculous meningitis is the sequel.

Even when examined grossly and microscopically the differential diagnosis between tuberculoma and gumma may be uncertain. The differentiation from coccidioidal granuloma may also present difficulties.

The *gumma* occurs as a late development of syphilis, but is rarely seen now. Formerly it was considered the process to be ruled out first and foremost in any case of suspected space-occupying lesion of the central nervous system. It is only a part of a more generalized disease process but may be the only manifestation thereof even to the extent of negative serological tests. Nonne[10] (1913) stated, "In general, one can think of the possibility of a gumma whenever the symptoms of an extra- or intramedullary tumor are present." Today the rare occurrence of a gumma in the spinal cord or its meninges has so altered the situation that gumma warrants only slight consideration in the differential diagnosis of neoplasm of the spinal cord.

In a large percentage of the few cases in which the central nervous system is involved, the gumma starts in the meninges and may encroach upon the substance of the brain and the spinal cord or may remain entirely extraparenchymal. The lesion is usually solitary but may be multiple and varies in size from 1 to 2 mm. to 4 or 5 or more cm.

In the spinal canal the extension of the tumefaction produces a fusiform enlargement of the cord and associated structures with fine adhesions

between the meninges and the cord proper. The mass and cord fill the dural sac, become unusually soft, and produce the signs of a true tumor of the spinal cord.[13] Extramedullary gummata form flattened masses which compress or encircle the spinal cord, giving rise to symptoms of compression, subarachnoid block and Froin's syndrome with its xanthochromia, high total protein, and spontaneous coagulation of the cerebrospinal fluid on standing. In other cases the meninges may be involved minimally, the process being limited to the nerve roots, along the course of which a number of small enlargements may be found.[10]

Coccidioidal granuloma in man is a chronic, progressive, often fatal fungus disease caused by the Oidium coccidioides (Coccidioides immitis). The central nervous system and meninges are often involved. Coccidioidosis was first reported by Wernicke[25] in 1892 and in the United States for the first time by Rixford[14] in 1894. The first report of coccidioidal infection of the central nervous system was in 1910 by Ryfkogel;[15] the patient was a child with coccidioidal meningitis. Dickson,[2] in 1915, described a coccidioidal destruction of the lumbar vertebrae and involvement of the paravertebral musculature, but it was not until 1930 that coccidioidal granuloma involving the spinal cord was reported. At that time Rand[12] reported 2 cases simulating true cord tumor. In 1 case the symptoms were those of compression at the level of the 6th thoracic vertebra with paraplegia and complete sensory loss below the 9th dorsal dermatome. Laminectomy revealed an extradural granuloma at the level of the 6th thoracic vertebra communicating through an opening between the 5th and 6th ribs, with a pocket of caseous material in the right chest. In the 2nd case there was a subdural granuloma completely encircling the cervical cord. In Rand's case the histological picture was very similar to that of tuberculosis with giant cells, lymphocytes, epithelioid cells and plasma cells, but the giant cells contained the double-contoured spherules of Coccidioides immitis. The presence of these parasites was the only reliable means of differentiation. In general, the disease may be primary in the skin, where it is usually confined as an ulceration which may become rather large. In other instances it may occur in the lungs and give the clinical picture of pulmonary tuberculosis, or in the skeletal system, again to be almost indistinguishable from tuberculosis. A diagnosis is made on the absence of tubercle bacilli, on microscopic examination, and the presence of the fungus. The spherules are scattered throughout the pathological tissues and many of them are taken up by the giant cells.[18] In some instances the fungus is surrounded by eosin-staining club-like processes to form a well defined pseudo-rosette.

Reports on torular infections of the central nervous system emphasize the meningitis produced by *Torula histolytica* and largely disregard the gross granulomas. That torular granulomas do occur in the central nervous system was shown by Smith and Crawford[16] in 1930 and by Goodhart and Davison[5] in 1937. In 1944 Swanson and Smith[20] reported 2 such granulomas simulating cerebral tumors. As for torular tumor of the spinal cord, as far as can be

ascertained only 2 cases have been reported. One of these (Smith and Crawford) involved the thoracic portion of the cord and the undersurface of the dura mater. The tumor measured $\frac{1}{4} \times \frac{1}{2} \times 1$ cm. Goodhart and Davison reported the 2nd case, an intramedullary granuloma.

The diagnosis, clinically, may be difficult, especially when the cerebrospinal fluid examination is negative in spite of a proven torular pulmonary disease. Whereas the spinal fluid may be entirely negative with the granuloma, in torular meningitis the organisms may be grown with ease even on routine culture media.

The gross appearance of the torular granuloma is quite characteristic, but at first glance it may resemble a glial tumor. Section reveals a multiloculated cyst, filled with a gelatinous material which does not empty itself when opened. In this way it is unlike a true cystic tumor and this is its most striking feature. The lesions may be multiple but single ones with findings resembling those of an isolated neoplasm do occur. There is a tendency to sharp demarcation from the adjacent structures and an apparent absence of reaction of the surrounding tissues. This constitutes another striking feature of the process. It would seem that surgical removal could be accomplished easily, but only rarely is there a true absence of an adjacent meningeal involvement. In addition, there is always the danger of opening the cyst and causing a rapidly spreading, purulent meningitis.[20]

No cases of intradural *pyogenic granuloma* were found in the literature reviewed, but the possibility is obvious. The same statement holds for noninfectious foreign body granulomas. Alajouanine and Petit-Dutaillis did report an epidural granuloma occurring in a French soldier 16 years after a bullet causing paraplegia had been removed from the spinal cord. With the development of the granuloma the soldier again developed a paraplegia. Warren and Romano,[22] Turnbull *et al.*,[21] and Watts and Mixter[23] have reported intraspinal pyogenic granulomas but all were epidural. One case of Watts and Mixter presented minor arachnoidal involvement but this was apparently secondary to the dural involvement. Watts and Mixter pointed out the accompanying fever and leucocytosis as aids in the diagnosis of pyogenic granuloma simulating cord tumor, but the other observers made no mention of these points.

Various observers have reported instances of granulomas occurring in the central nervous system as the result of infestation with *schistosoma*.[17] This is primarily a disease of the Far East and occurs in the Occident only in persons who have recently resided in the Orient, as with the former soldiers reported by Hunt, Abramson and Weaver,[7] and by Swanson.[19]

Of the three varieties of schistosoma, S. japonicum involves the central nervous system much more frequently than do the others. This type is endemic in Japan, China, Formosa, the Philippines and the Celebes. Neurological complications caused by S. hematobium are extremely rare. This type is found exclusively in Africa (particularly Egypt) and causes urinary schistosomiasis. The third variant, S. mansoni, prevails in Africa, the West

Indies and northern South America. Instances of involvement of the central nervous system by this parasite are even more rare. It is said to favor the spinal cord.[7] Müller and Stender presented a case of transverse myelitis of the dorsolumbar cord caused by S. mansoni.[9] Gama and Marques de Sá[4] reported a case of granuloma of the spinal cord caused by S. mansoni. There was a rapidly developing paraplegia in an otherwise healthy man of 42 years. A laminectomy revealed the true nature of the lesion, which was shown microscopically to be a granuloma containing the ova of S. mansoni. The past history of the patient revealed episodes of other symptoms suggestive of schistosoma infestation.

The possibility of this disease must be considered in the case of former service men or former residents of areas where the infestation is endemic. Episodes of other symptoms suggestive of such an infestation (rash, fever, diarrhea, abdominal cramps, stiff neck, changes in sensorium, muscular weakness, convulsive seizures, eosinophilia, positive intradermal tests and parasitic ova in the stools) may be of help in establishing the diagnosis. These findings, in all probability, will be of more value in retrospect than in foresight.

The cerebrospinal fluid findings are inconsistent, varying from normal to findings of slightly lowered sugar content, moderately elevated total protein, pleocytosis of slight degree and abnormal colloidal gold curves of the paretic type. The stool examinations may be negative for the ova of schistosoma. Eosinophilia may be absent.

Grossly, the granuloma varies in color from red purple to yellowish-gray and at times may resemble a glioblastoma multiforme. It may be of firm consistency and again may be quite soft. Microscopically, giant cells abound and contain the ova of the parasite. Plasma cells, lymphocytes and eosinophiles are found at the periphery. The center may be necrotic. The lesion may be a single granuloma or a coalescence of many smaller granulomas.

Surgical removal, if possible, should be followed by intensive therapy with the antimony derivatives, such as tartar emetic or Fuadin.

There are no clinical manifestations characteristic of compression of the spinal cord by granulomas that can be depended upon to differentiate them from those produced by a true neoplasm in the same location. To some degree, the compressive symptoms as produced by the granuloma may be overshadowed by manifestations of the myelitis or meningitis which may precede or accompany the granuloma. Again the possibility of a granuloma might be considered when evidence exists of a generalized disease process which might give rise to granulomatous formations. The symptoms vary greatly with the level involved and with the relation of the mass to the spinal cord, spinal nerve roots and meninges, as is obvious. Symptoms typical of a cauda equina tumor can be produced as shown by Ray[13] who, in 1940, reported a case of gumma simulating such a lesion.

In the extramedullary location, the first symptoms will probably be those of an irritative sensory nature because of involvement of the posterior

roots. Eventually the complete picture of compression of the spinal cord with anesthesia and paralysis below the level of the lesion, alterations in the reflexes and in the function of the bowel and bladder will develop.

The granulomas themselves offer definite differential diagnostic problems, even when examined grossly and microscopically. A selective diagnosis of gumma, tuberculoma or other granuloma will often rest upon evidence or lack of evidence of one or another disease process elsewhere in the body.

Since the differential diagnosis between granuloma and neoplasm is virtually impossible clinically, the therapeutic course, which should always be followed closely, is obvious. To subject the luetic patient, for example, to a course of antiluetic therapy before considering operation is not wise for two reasons. First, the lesion may be neoplastic and thus not amenable to such therapy. Second, even though it be a gumma it may not respond to such treatment, while in the meantime the spinal cord will be further and possibly irreparably damaged by compression. The first step should always be laminectomy and decompression of the lesion and spinal cord. Later, antiluetic or other specific or supportive therapy may be administered.

Although, in the main, the prognosis can not be regarded as good with a group of diseases that are typified by widespread involvement of the body and for which (with the exception of syphilis) there is no specific therapy of dependable value, the case of intradural granuloma of the spinal cord reported here is incontrovertible evidence that adequate decompression of the cord by laminectomy and partial removal of the granuloma may, on occasion, permit the body to overcome the infection and go on to a satisfactory readjustment and clinical recovery from the disease.

The following case is unusual and of interest for several reasons. In this patient an intradural granuloma developed in intimate relation to the leptomeninges and the spinal cord. It was apparently tuberculous in origin although no other loci of tuberculous infection have been demonstrated. Most surprising of all, its partial removal has been followed by a complete recovery of the patient from all of the symptoms of involvement or compression of the spinal cord. In the 3 years that have elapsed since her operation there has been no evidence of extension of the lesion or of the infection. Specifically, a tuberculous meningitis did not develop.

CASE REPORT

L.O., a 44-year-old widow, referred by Dr. F. L. Jenkins of Chicago, was admitted to the Chicago Memorial Hospital on Feb. 6, 1946. She had been in good health until July, 1945, when she arose one morning with a "toothache-like pain" in the lower thoracic and upper lumbar region of her back. The discomfort lasted all morning and then disappeared, only to recur about 1 hour later to last for the rest of the day. This was a daily cycle for 1 week, after which the pain left her and never recurred. There were no accompanying symptoms.

Following this brief episode she was well until mid-November, 1945, when she noticed "numbness" and coldness of her feet. This peculiar feeling progressed upward

slowly and was accompanied about 3 weeks later by weakness of the legs. The weakness gradually progressed and was more marked in the right leg. Soon she required a cane and by Jan. 31, 1946, she became unable to stand or walk.

In late November, 1945, a band of burning sensation developed at the level of the umbilicus with radiation to the right and left around to the back. The pain was constant and was aggravated by twisting of the trunk. She denied having any dysfunction of bowel or bladder, but a uriniferous odor was noted during the examination. She denied ever having had syphilis or gonorrhea.

Examination. There was a slight bloody discharge from the cervical os, which showed minimal erosion. There was slight dorsal kyphosis.

On neurological examination the cranial nerves were entirely negative, as were the upper extremities. Hypalgesia and hypothermaesthesia were present bilaterally below the 10th thoracic dermatome. Vibratory sensibility was absent below the 2nd

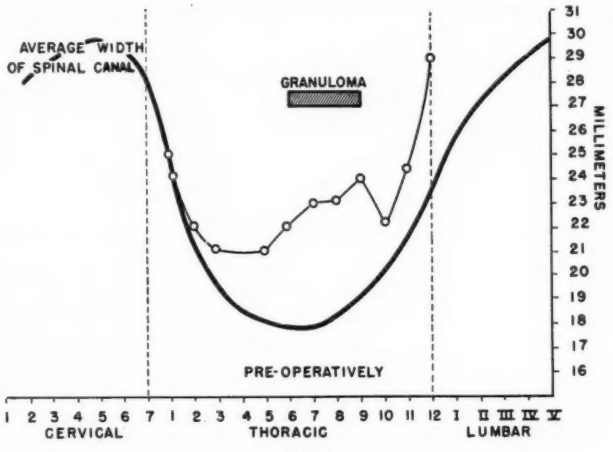

FIG. 1

lumbar spine. Position and localization sense and two-point discrimination were normal in the upper extremities but absent in the lower. Deep pain sense in the tendo achillis was present. The lower extremities, particularly on the right, showed definite weakness in all movements and were spastic. Heel-to-knee tests were impossible. Beevor's sign was positive. Knee jerks were hyperactive, the right more so than the left. Ankle jerks were absent, as were abdominal reflexes (obese, flabby, abdominal wall). Babinski, Chaddock, Gordon and Gonda signs were all positive bilaterally. There was no nuchal rigidity. Kernig, Lasègue, Patrick and Naffziger tests were negative.

Lumbar puncture in the 4th interspace revealed an initial pressure of 150 mm. of fluid and an almost complete block of CSF flow on bilateral jugular compression. Abdominal compression caused a rapid rise to over 200 mm. with a rapid fall following release. Four cc. of xanthochromic fluid were removed: there were 5 rbc/c.mm.; Pandy test 4+; total protein, 6800 mg.

Roentgenograms of the chest were not unusual. The thoracic spine showed a minimal degree of osteoarthritis. Measurements of the interpedicular spaces showed a widening of the spinal canal at the level of the 5th, 6th, 7th and 8th thoracic

vertebrae (Fig. 1). The pedicles otherwise appeared normal with no apparent evidence of atrophy or erosion.

A diagnosis was made of extramedullary tumor within the spinal canal causing compression of the spinal cord at the level of the 6th to the 8th thoracic vertebrae.

Operation. On Feb. 8, 1946, a laminectomy was performed. The dura mater bulged backward with the apex at the level of the 7th thoracic vertebra. On palpation a firm mass was found at this level. The dura mater appeared normal. Immediately beneath it was a grayish, avascular mass which was attached to the dura mater at the level of the 7th thoracic vertebra, but extended upward to the top of the 6th thoracic vertebra and downward to the lower limits of the 8th. This mass, however, was largest in the middle. It covered the spinal cord completely. At the upper limits the mass merged imperceptibly with a somewhat thickened, opaque arachnoid membrane. Looking up the spinal canal beneath the 5th thoracic vertebra one could observe an irregularly thickened, milky arachnoid membrane as far as it was possible to see. When this was incised, the spinal cord and pia mater appeared normal. The mass arose from the arachnoid membrane and was much thicker and larger on the right side. An incision was made through the arachnoid membrane along the posterolateral part of the spinal cord on the left side. The mass was then reflected toward the right off the spinal cord, to which it was attached by numerous dense adhesions which had to be divided by sharp dissection. As this dissection proceeded around the right side, the mass became more and more densely adherent to the cord, which was markedly compressed, particularly from the right side (Fig. 2). All of the mass lying posterior and to the right of the spinal cord was removed. It was obvious, however, that there was still some of the mass lying anterior to the cord, which was pushed backward sharply at the level of the 7th thoracic vertebra. This part of the mass was left in place as it could not be removed without seriously endangering the already compressed and contused spinal cord.

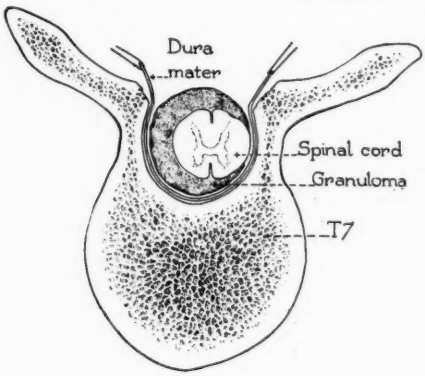

FIG. 2.

A frozen section revealed a granuloma of unknown etiology. A piece of tumor was taken for culture and the remainder sent to the laboratory for microscopic study. The usual closure, including closure of the dura mater, was then effected.

Laboratory Studies. On Feb. 9, 1946, the blood serological findings were reported as Wassermann negative, Kahn positive and Eagle weakly positive. The CSF Wassermann was negative and the Lange colloidal gold curve was 0000122100. A second examination of the blood showed Wassermann and Kahn tests both negative. Still another examination was again negative. Similar tests previously made were also negative. During operation a sample of CSF had been taken from the subarachnoid space above the point of obstruction; Wassermann test on this was negative and Lange was 0000122000.

Postoperative Course. There was very rapid recovery of motion of the lower extremities, but rather slower recovery of position and vibratory sense. By the 6th postoperative day she was moving both legs well; position sense was still very poor

but vibratory sensibility was excellent. The patient was able to extend and flex the legs well at both hips and knees. On the 12th postoperative day she sat up with help, and on the 14th day she was discharged to her home.

Antiluetic therapy was then started by Dr. F. L. Jenkins pending further investigation of the true nature of the granuloma. Therapy was discontinued when the pathologist's final report was received.

Pathological Report (Dr. Otto Saphir). There was a diffuse granulomatous reaction characterized by scattered small foci of necrosis surrounded by epithelioid cells. Numerous multinucleated cells of the Langhans type were noted and lymphocytes

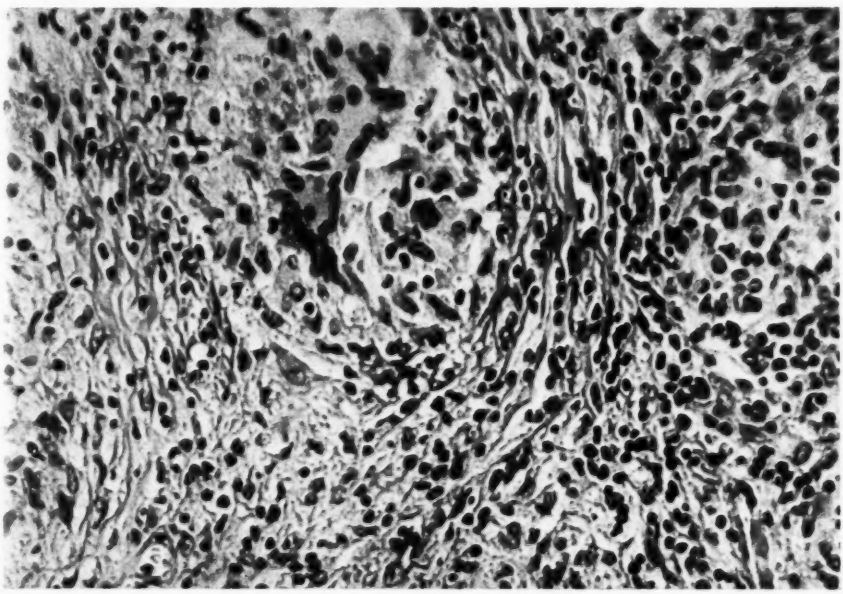

FIG. 3. Photomicrograph of granuloma in authors' case.

were abundantly interspersed throughout the section. Strands of hyaline and fibrous tissue bordered the section, and more fibrous tissue was noted between the granulomatous areas. Except for several vascular areas in one region, blood vessels were very sparse (Fig. 3). Ziehl-Nielsen staining failed to reveal any tubercle bacilli. Impression: Tuberculous granulation tissue.

Bacteriological Report. Smears made for acid-fast organisms were negative; 6 weeks postoperatively the laboratory reported a growth, on culture, of colonies of acid-fast organisms, which colonies the bacteriologist stated were not typically those of the tubercle bacillus. These organisms were then inoculated into a guinea pig. Later, on May 2, 1946, the laboratory reported that the guinea pig had died and on autopsy was shown to have many typical tuberculous lesions. This diagnosis was confirmed by Dr. Otto Saphir, the pathologist.

Course. The patient showed gradual improvement. When seen on Mar. 18, 1947, she walked without assistance. She stated that from the time of her discharge she had no trouble with her bladder and only occasionally had required an enema. She

began to be up and about on crutches in May, 1946, and began to walk unaided by mid-July, 1946. She pointed out the right 10th thoracic dermatome as the site of occasional burning pain which lasted a day or two. She was able to walk at least two miles with no difficulty.

Neurological examination showed a band of hypalgesia at the 9th thoracic dermatome on either side but sensibility was otherwise normal everywhere. A slight weakness in flexion of the left knee was present. The abdominal reflexes again were not elicited because of a thick abdominal wall. Beevor's sign was negative. The right knee jerk was slightly more active than the left and the ankle jerks were absent bilaterally. The Babinski and other signs showed a normal plantar response.

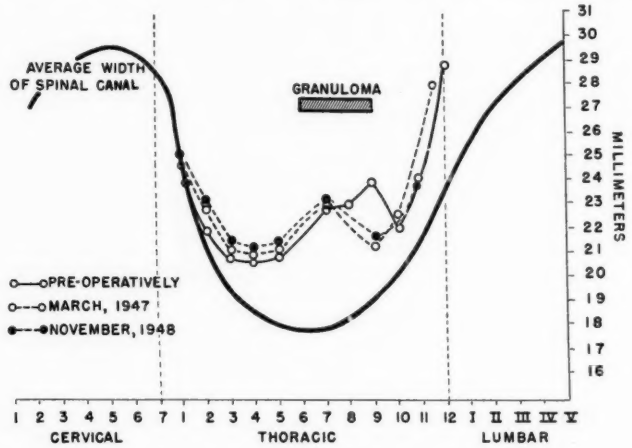

FIG. 4

Her gait was slightly wider based than normal and the left leg was favored ever so slightly. Romberg's test was negative.

X-ray examination of the thoracic spine showed the laminectomy and increase in the density of the pedicles at the site of the former tumor. The interpedicular distance was decreased as compared to the preoperative measurements (Fig. 4). A blood Wassermann and Kahn, as well as urinalysis and blood count, all proved entirely negative. No lumbar puncture was made.

On Nov. 15, 1948, at the time of her last examination, she stated that she had had no difficulties whatever. Gait and station were entirely normal. No muscular weakness was demonstrated. The tendon reflexes were equal bilaterally and no pathological reflexes were found. The sensory examination was likewise negative.

Roentgenograms of the thoracic spine centering on the 7th vertebra showed no essential differences from the previous interpedicular measurements of March, 1947 (Fig. 4), but the pedicles themselves appeared better calcified and more convex on their medial surface. No lumbar puncture nor serological blood study was made.

Inquiry several months later disclosed that the patient had worked as a kitchen helper and waitress at a tuberculosis sanatorium some 10 years prior to the onset of her symptoms. In her work she had contact with the ambulatory patients who had their meals served in the dining room of the sanatorium. Frequent chest examinations, including roentgenograms, were made on all employees of that institution

but the patient was never found to have any objective evidence of tuberculosis. Neither did she have any subjective symptoms. Inquiry as to social or family tubercular contact was negative. She has always lived in Iowa or in Illinois and has never left these states.

COMMENT

There are several confusing points about this interesting and unusual case. In general, the symptoms and findings are clearly explained by the lesion, except for one point. We do not understand why this lesion at the level of the 6th to 8th thoracic vertebrae, which gave rise to hyperactive knee jerks and Babinski's sign, was associated with absent ankle jerks.

Apparently the one examination of the blood in which the Kahn test was positive and the Eagle test weakly positive was false and misleading, as all other examinations for syphilis were repeatedly negative. It seems, therefore, unlikely that the antiluetic therapy begun postoperatively before it was finally concluded that the lesion was not luetic in origin played any role in the patient's recovery.

If, as finally seemed most likely, the lesion is tuberculous in origin, there are several facts that are difficult to understand. It should be recalled that the granuloma was only partially removed, that it lay intradurally and in intimate contact with the leptomeninx and the spinal cord. In such circumstances, one would ordinarily expect a fatal tuberculous meningitis to develop shortly. Here, on the contrary, not only did no meningitis appear, but the patient made a complete recovery which has been sustained now, some 3 years after the operation. We have no explanation for these facts. If we refuse to accept the tubercle bacillus as the responsible agent we have no grounds for acceptance of any of the other less common causes of granuloma of the central nervous system. Furthermore, in spite of the unusual aspects of the case, a tuberculous infection is by all odds the most likely for the following reasons: (1) Tuberculosis is the most common cause of a granuloma of the central nervous system in a resident of the central United States. (2) This patient had had extensive exposure to tuberculosis. (3) The tissue was diagnosed as a tuberculous granuloma on the basis of its microscopic appearance. (4) Acid-fast organisms were grown in cultures of the tissue removed at operation. (5) These organisms produced an acute disseminated disease in guinea pigs which was diagnosed as being tubercular.

SUMMARY

A case of intradural spinal granuloma is presented. The patient had a gradually progressive paraparesis with complete obstruction to the flow of cerebrospinal fluid and greatly increased cerebrospinal fluid total proteins. Laminectomy revealed a granuloma compressing the thoracic spinal cord. This was partially removed and a complete recovery effected. Later investigation proved almost certainly that this was a tuberculoma.

The various types of granulomas occurring subdurally in the central nervous system and especially in the spinal cord are discussed.

The difficulty of differentiation of granulomas from tumors is stressed.

It is recommended that the lesion, in cases where the spinal cord is

involved by a granuloma, should be exposed by a laminectomy and as thoroughly removed as possible. Systemic treatment should not delay the decompression of the spinal cord but where such specific treatment is possible it should be carried out postoperatively.

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SURGICAL TREATMENT OF ANEURYSM OF THE ANTERIOR CEREBRAL AND OF THE ANTERIOR COMMUNICATING ARTERIES DIAGNOSED BY ANGIOGRAPHY AND ELECTRO-ENCEPHALOGRAPHY*

ARTHUR R. ELVIDGE, M.D., AND WILLIAM H. FEINDEL, M.D.
Department of Neurology and Neurosurgery, McGill University, and the Montreal Neurological Institute, Montreal, Canada

(Received for publication April 28, 1949)

ALTHOUGH the subject of intracranial aneurysm has received too much attention in recent neurosurgical literature to consider it any longer "a lesion having such remote surgical bearings" (Cushing[5]), nevertheless it continues to pose many difficulties in diagnosis and treatment. Many of the diagnostic problems have been clarified by the stimulating clinical studies of Symonds,[51] and of more recent investigators including Albright,[1] Strauss, Globus and Ginsburg,[49] Bramwell,[2] McDonald and Korb,[32] Richardson and Hyland,[41] and Jefferson,[22] and particularly by the development of intracerebral angiography by Moniz,[35] Dott,[8,18] Jefferson,[23] Elvidge,[11] Engeset,[12] and others.

The problems related to the surgical therapy have been reviewed by Dott,[9,10] Jefferson,[24] Matas,[34] Krayenbühl,[26] Dandy,[7] List and Hodges,[30] Gross[15] and Poppen.[38] The rôle of carotid ligation in treatment has been discussed by Schorstein,[47] Dandy,[7] Olivecrona,[36] Krayenbühl,[27] Dott[10] and Jefferson.[24]

Relatively few instances of successful surgical treatment of aneurysms located on the anterior cerebral or anterior communicating arteries have appeared in the literature. The present communication is concerned with 2 cases diagnosed by angiography and treated by direct surgical exposure. Follow-up now extends over 3 years.

REVIEW OF LITERATURE

Carotid Ligation. In the cases previously published, treatment has been carried out by carotid ligation or by direct exposure of the aneurysm or occasionally by a combination of these two methods. Most saccular aneurysms for which carotid ligation has been performed have been situated on the carotid artery itself, but several authors mention anterior cerebral aneurysms successfully treated by this method.

Krayenbühl[28] has now ligatured either the common or internal carotid in 8 cases of bleeding anterior cerebral aneurysm. One patient recovered from a bitemporal hemianopsia caused by a large aneurysm of the anterior communicating artery.[27] In another case of severe haemorrhage from an

* Read in part at the thirty-sixth meeting of the Society of British Neurological Surgeons, Lisbon, April 10, 1947, and the American Academy of Neurosurgery, Colorado Springs, October 11, 1947.

aneurysm of the left anterior cerebral artery satisfactory recovery followed ligation of the left common and internal carotid arteries.[27] Olivecrona[36] reported 9 cases of carotid ligation for supraclinoid saccular aneurysm, and 2 of these, noted as anterior cerebral in location, were improved by carotid occlusion. In a patient who had had two attacks of haemorrhage, 2 weeks apart, with hemiparesis from an aneurysm at the junction of the right anterior cerebral and anterior communicating arteries, Walsh and Love[53] ligated the right internal carotid artery. Except for two attacks of "syncope," neurological examination was reported to be normal at the end of 3 months.

Results in these cases suggest that carotid ligation may be effective in the prevention of recurrent bleeding from aneurysms not only of the carotid artery but also those of the main cerebral vessels. The local lesion, however, still remains and its behaviour over a long period is not predictable. A larger series of cases with longer periods of follow-up will be necessary before the value of this method can be properly assessed.

Intracranial Approach. The method of direct intracranial exposure, albeit hazardous, is more precise. Nine cases of anterior cerebral aneurysm treated successfully in this manner have been reported. In 2, the aneurysm was excised; in 1, trapped between clips, and in the remaining 6, pieces of muscle were placed around or inside the aneurysmal sac. The recoveries are perhaps the more remarkable considering that in only 2 cases was a definite diagnosis by angiography established, and, in addition, the sac ruptured during operative manipulation in 4 instances.

The earliest reported case (Dott[8]) was that of a man aged 53 years with an aneurysm at the junction of the left anterior cerebral and the middle cerebral arteries. The sac ruptured during the operation. Haemorrhage was controlled by muscle tamponade. The patient was well 6 years following operation.

Tönnis[52] demonstrated by arteriography an aneurysm of the anterior communicating artery. He exposed it by splitting the genu of the corpus callosum. Blood clot was removed and the sac surrounded with bits of muscle. The patient returned to work and was well a year later.

A leaking aneurysm of the right anterior cerebral artery in a boy aged 12 was removed by Cone in 1938 and reported by Russel.[46] An encephalogram showed evidence of an expanding lesion in the right frontal region. An intracerebral haematoma was evacuated. This revealed an aneurysm, which was clipped and excised. The patient was in good health $7\frac{1}{2}$ years later.[4] This is the first recorded case of successful excision of an intracranial arterial aneurysm.

Dandy[7] reported 2 successful results in 6 cases of anterior cerebral aneurysm. In his Case XXIV, a man of 49 years operated upon in 1941, the lesion, suspected of being a neoplasm, was a huge mass lying below and in front of the optic chiasm. It proved to be an aneurysm and was thought to arise from the anterior cerebral or the anterior communicating artery. The sac was opened, packed with muscle and cauterized. The left internal carotid artery

was partially occluded 7 days later with a band of fascia and totally occluded in another 10 days. The patient's pre-operative aphasia, right motor weakness, loss of memory, personality changes and headaches disappeared.* He returned to work and was well 13 months after operation.

In Case XXV (Dandy[6,7]), a woman aged 45 years, clinical signs suggested the diagnosis of intracranial tumor. An aneurysm was disclosed arising from the anterior cerebral artery, and ruptured during manipulation. Haemorrhage was controlled by clipping the intracranial portion of the internal carotid artery. The aneurysm was excised and included resection of the upper half of the right optic nerve. The anterior portion of the softened left frontal lobe was removed at a later operation because of the development of drowsiness and postoperative seizures. The patient was well 13 months after operation. Klemme and Woolsey[25] described a case in which a hard non-pulsating mass under the left optic nerve yielded a gush of blood on curettage and a strip of muscle was inserted into the sac, which was considered to arise from the anterior cerebral artery. The patient recovered except for the pre-operative visual defect, and showed no evidence of recurrence for 4 months.

Jefferson[22] mentions a case in which an anterior cerebral aneurysm was packed with muscle. Symptoms improved but fatal rupture occurred 18 months later. List and Hodges[30] successfully trapped by clips a small aneurysm of the left anterior cerebral artery. A case that might be mentioned is one of subchiasmal aneurysm which simulated clinically a pituitary tumour (McConnell[31]). The sac was opened and packed with muscle. The patient showed improvement of vision and remained without recurrence for several years.

CASE REPORTS

Case 1. Neurosurg. No. 12809. Recurrent attacks of headache and vomiting for 2 years. Intracranial haemorrhage with sudden onset of severe headache, aphasia, stupor, and development of right hemiparesis. Aneurysm at junction of anterior communicating and left anterior cerebral arteries disclosed by thorotrast arteriography. Surgical exposure and clipping of aneurysm. Recovery. Follow-up for 3½ years.

M.P., male, aged 20, was admitted to the Montreal Neurological Institute in a confused state on Sept. 24, 1945. For 2 years he had had attacks of severe frontal headache sometimes associated with vomiting. These recurred 5 times in a year and lasted some 12 hours; the patient would stop work, go home to a darkened room and sleep. He had been "nervous" since childhood, but during the last year he had been more restless and nervous. On the evening before admission his father found him in bed complaining of severe occipital headache. When his head was lifted from the pillow the patient screamed with pain. During the night he vomited, was incontinent and appeared confused and irritable. He had attempted to put on ski boots although it was much too early in the season. By the next morning he was

* In Table C (XXIV) it is stated that the partial ligation took place on the following day and complete ligation 7 days later. It also states that his condition was unchanged on discharge 3 weeks after 1st operation.

considerably worse, showed no response to questions and was in a condition of agitated stupor.

Examination. He was a youth of slight build who exhibited marked somnolence, but nevertheless was restless and agitated. There was evidence of meningeal irritation, bilateral Babinski signs and small pupils unreactive to light. He appeared to

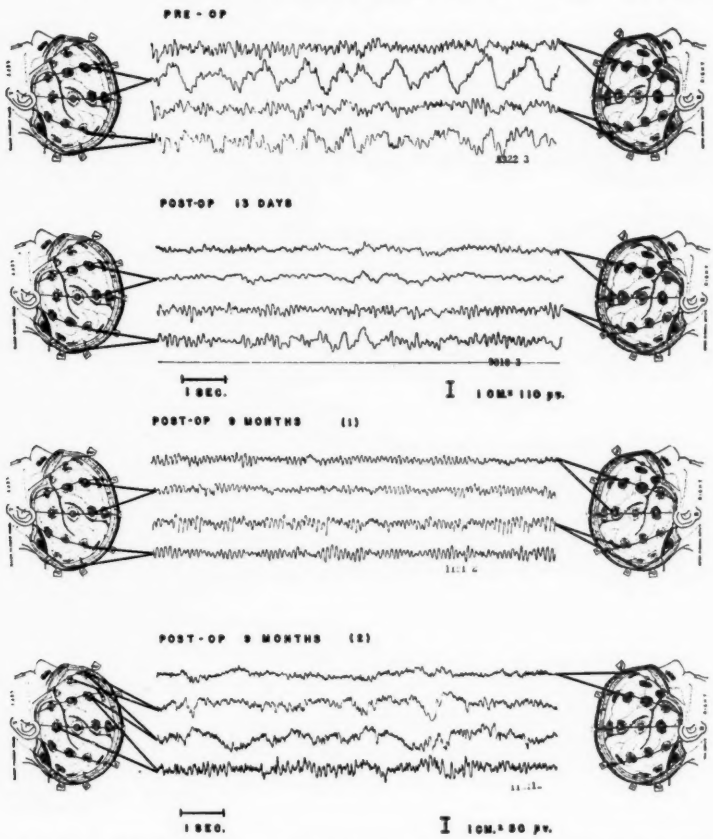

FIG. 1. Samples of pre-operative and postoperative EEG records in Case 1. Note the "pulsation artefact" in the pre-operative record (see text).

understand but gave no verbal response. Blood pressure was 110/40. CSF pressure was 350 mm.; the fluid was grossly bloody. There was little to suggest lateralization of the haemorrhage.

Over the next 2 weeks, in spite of clearing of the CSF, somnolence increased and was accompanied by a high fever, leucocyte count of 13,300/c.mm. and sedimentation rate of 46 at 1 hour, but no evidence of an infective process could be found clinically.

At the end of the 1st week a complete paralysis of the arm and weakness of the

leg on the right side developed. Right plantar was extensor; the left, equivocal. CSF, Wassermann and blood Kahn were negative. The prothrombin time was increased (62 sec.) and platelet counts reduced (180,000).

Electroencephalogram, Oct. 3, 1945, 4 days after onset of the right hemiparesis, showed large slow waves, fairly well restricted to the left frontotemporal region. A 2nd EEG, 2 weeks later, was similar. Rhythmic large slow waves from the left

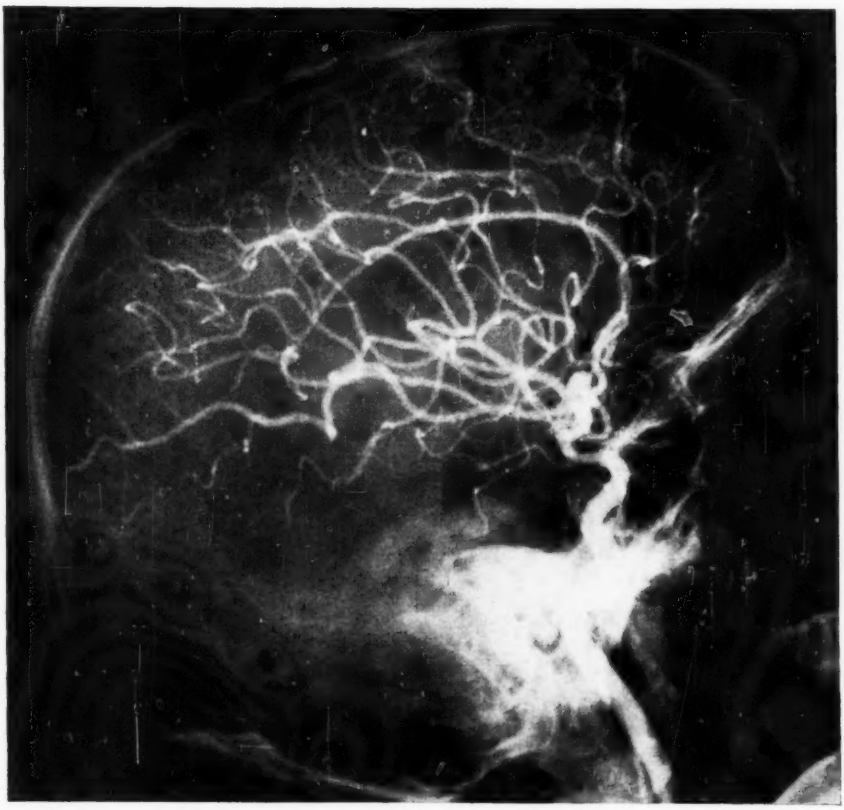

FIG. 2. First film of arteriogram in Case 1, with thorotrast. Arrow indicates aneurysm at junction of left anterior cerebral and anterior communicating arteries.

frontotemporal region at a rate coinciding with the pulse, probably represented a pulse artefact (Fig. 1, Pre-op.).

Angiography, Oct. 18, 1945. Thorotrast was injected into the exposed left common carotid artery in two successive amounts for lateral stereograms without changing syringe or needle and with the external carotid occluded by traction ligature.

The arteriogram showed a small aneurysmal dilatation 5 mm. in diameter which protruded (Fig. 2) from the superior surface of the anterior communicating artery.

The retention of thorotrast commonly seen within an aneurysm is shown on the phlebogram (Fig. 3) taken 4 sec. later.

Operation, Oct. 24, 1945. A left transfrontal osteoplastic craniotomy was made, the frontal lobe elevated, and the optic chiasm exposed. A loose ligature was placed around the left internal carotid artery intracranially as a safety measure. A lumbar puncture needle had been left in place in the spine in order to reduce brain volume

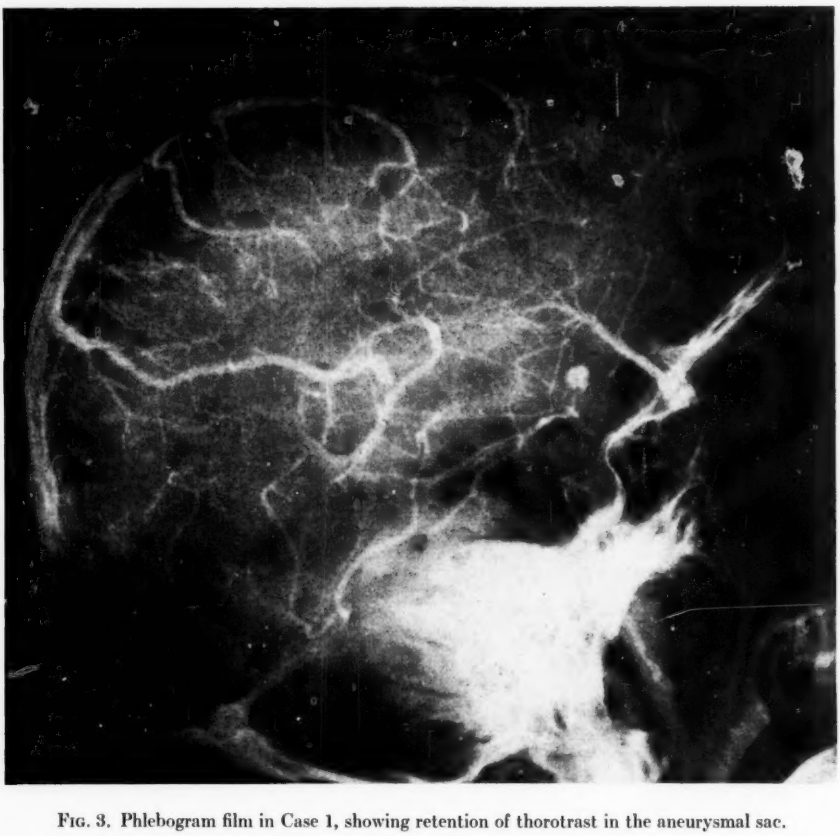

FIG. 3. Phlebogram film in Case 1, showing retention of thorotrast in the aneurysmal sac.

during the procedure. The bifurcation of the internal carotid into the middle and anterior cerebral artery was identified. The junction of the anterior cerebral with the anterior communicating artery was visualized; the anterior was followed some distance as it passed forward to the right, and the anterior communicating artery was traced to approximately the point of its junction with the opposite right anterior cerebral. The middle cerebral artery was seen to be normal for a distance of 1–2 cm.

A small pea-sized yellowish aneurysm was discovered pointing posteriorly from

the anterior communicating artery near its junction with the left anterior cerebral artery, exactly as was depicted in the angiographic films. Adjacent there was a small black and yellow discoloured area with some pia-arachnoidal adhesions, the only trace of former rupture.

A tantalum clip was placed on the anterior cerebral artery proximal to its junction with the anterior communicating artery and two clips distal to it as the anterior cerebral swung across the inferior medial surface of the hemisphere. Tantalum clips were placed on either side of the sac on the anterior communicating artery and finally one clip was placed over the aneurysm itself, obliterating its neck. Altogether six tantalum clips were used in trapping the aneurysm (Fig. 4). Lack of evidence of local haemorrhage is not surprising in view of the complete disappearance of blood from the CSF specimens 2 weeks before operation. No bleeding occurred from the

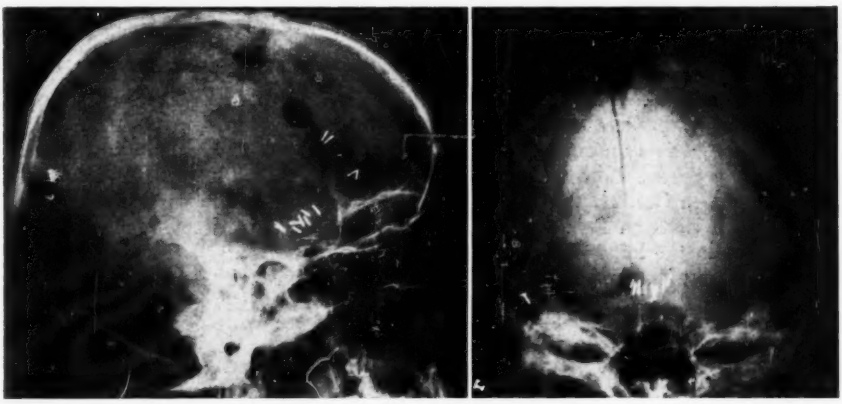

FIG. 4. Postoperative roentgenograms show position of tantalum clips, Case 1.

aneurysm during manipulation so that it was fortunately not necessary to use the ligature around the carotid in the middle fossa.

Postoperative Notes. His course was one of progressive improvement. He spoke for the first time since his hospital admission on the 2nd postoperative day and was talking freely 2 weeks later. He had occasional incontinence for 3 weeks. In the EEG 13 days after operation (Fig. 1) the amount of slow wave discharge from the left frontotemporal region was somewhat less and there was absence of the "pulsation artefact" previously recorded. He left hospital 25 days after operation.

Subsequent Course. On Feb. 12, 1946, $3\frac{1}{2}$ months postoperatively, the patient had no headache or dizziness. He could count rapidly in French and in English. Sense of smell was absent. His right leg appeared slightly stiff when he walked.

On June 4, 1946, there was merely a residual slight weakness of the right foot. Nervousness and fatigue were complained of and the patient did not go out due to lack of confidence in walking.

The EEG on July 16, 1946 (Fig. 1) demonstrated localized slow and sharp wave disturbance of moderate degree of severity, maximal in the left anterofrontal region, and in lesser amount from the left temporal region and from the homologous right side. The background activity from the parieto-occipital region was of good

amplitude and continuity, and fairly well regulated at about 10/sec. The persistence of these abnormalities was not surprising when one considers that the left anterior cerebral artery was occluded at operation. Neither the clinical signs nor the EEG were as severe as might have been expected in view of the serious consequences of this procedure that have been discussed (Dandy,[7] Poppen[37]).

On Aug. 6, 1946, the patient still exhibited slight stiffness of the right leg. He lacked initiative, would not go out, apparently as a result of fear, and did not associate with any of his friends. On Sept. 30, 1946, he stated that he could not go back to work because he could not write well. He meant that he could not write as fast as before. Actually he could write very well.

The EEG on April 11, 1947 demonstrated a persistent area of very slow delta activity localized to the pole of the left frontal lobe. The delta activity was not widespread as previously, which suggested progressive improvement, but indicated a residual area of damaged cerebral tissue in the anterior pole of the left frontal lobe.

When tested in April 1947 sense of smell was intact. On May 2 the patient had an epileptic seizure with loss of consciousness and clonic movements in the extremities while asleep. This attack was followed by vomiting. When seen a month later and again in October 1947, he was still fearful of going out alone. His right hand was objectively normal but he still complained that he could not write as fast as before his illness. He walked well but his toe still rotated inward.

An EEG on Oct. 31, 1947 showed no further change and in particular no spike activity. An EEG on Feb. 5, 1948 showed slight improvement with residual slow wave focus in the left frontal pole. On July 20, 1948 EEG showed well regulated alpha rhythm with slow waves present but not prominent. Sharp waves in the left frontotemporal region suggested the presence of a potentially epileptogenic lesion.

When last seen in April 1949 the patient was very well. He exhibited the slightest residual internal rotation of the foot on walking. He was willing to work if he could be given a position without too much writing. His original occupation called for a great deal of close application.

Case 2. Neurosurg. No. 4938 and 6371. Sudden collapse from subarachnoid and intracerebral haemorrhage, localized clinically to left frontal region. Improvement. Discharge from hospital 1 month later with recurrence the same day and readmission. Diodrast angiography demonstrating aneurysm of right anterior cerebral artery. Excision of aneurysm and recovery. Clinical and EEG follow-up for 3 years.

History. R.O., aged 13, stated that he was struck on the head by a hockey stick, on April 8, 1946. He was taken to a local hospital where a lumbar puncture showed blood in the CSF. He became drowsy, irritable and incontinent.

Examination. On admission to the Montreal Neurological Institute 2 days later, patient exhibited stiffness of the neck, positive Kernig and positive Babinski sign, more complete on the right. The right pupil was larger than the left and there was paralysis of the left abducens nerve. Blood pressure was 130/90; pulse rate 60; temperature 100.8°F. Lumbar puncture showed grossly bloody fluid under normal pressure. The following morning weakness of the right leg developed, more noticeable in the distal groups of muscles, with a definite plantar extension on the right and questionable extension on the left. Pupils were normal but the left 6th nerve paralysis remained. The fundi showed fulness of the veins but no elevation of the discs. X-ray films of the skull were normal. Further lumbar punctures showed normal pressure and yielded yellow CSF.

EEG, recorded 2 days after onset of illness, showed severe generalized disturbance with slow waves and absence of alpha rhythm, but no evidence of a focal lesion (Fig. 5, Pre-op. 1).

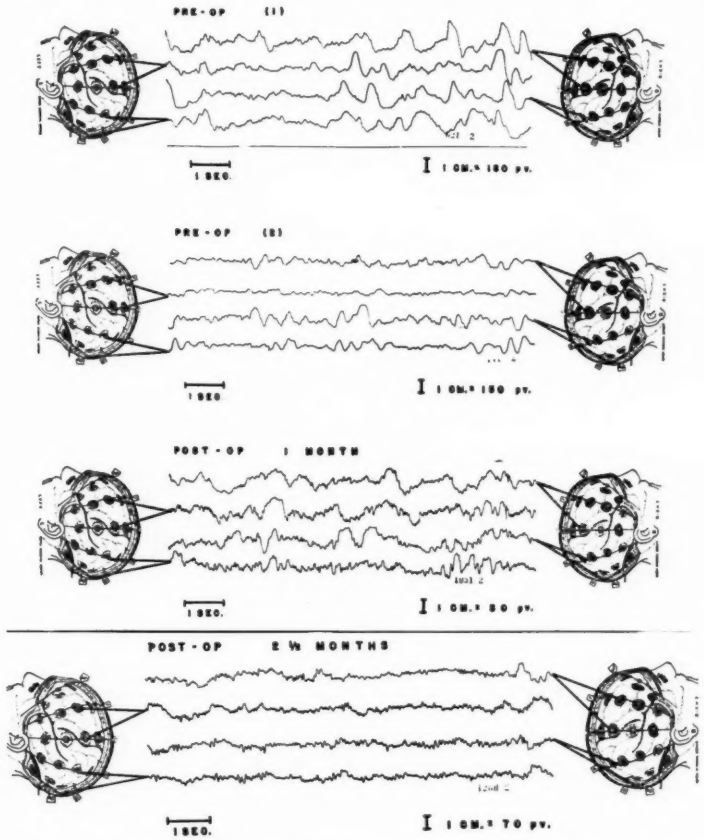

FIG. 5. Samples of pre-operative and postoperative EEG records in Case 2 (see text).

Pneumoencephalogram on April 20 showed relative absence of subarachnoid markings over the frontal poles, especially on the left; dilated lateral and 3rd ventricles, and evidence that was interpreted as indicating that a subarachnoid haemorrhage in the lower left frontal region had communicated with the anterior horn of the left lateral ventricle (Fig. 6). An EEG repeated on April 30 (Fig. 5, Pre-op. 2), indicated improvement by an increase of wave frequency, but there remained severe delta wave disturbance, most marked in the *right* temporoparietal region and suggesting a deep-seated lesion. Normal alpha rhythm was not observed.

The boy showed progressive improvement with almost complete recovery except for the left abducens paralysis and at the end of 4 weeks was discharged from hospi-

tal with the diagnosis of subarachnoid haemorrhage probably due to ruptured intracranial aneurysm.

Readmission. On the day of his arrival home, May 6, 1946, he suffered a severe pain in the head while straining at stool and his right leg and arm started to shake. He was readmitted to hospital where he appeared drowsy and complained of severe headache. There was bilateral plantar extension. Pupils were sluggishly reactive to light and disc margins were hazy. The left 6th nerve paresis was still present and lumbar puncture gave bloody fluid. During the next 2 weeks he complained of headache and dizziness and moderate papilloedema developed. The Babinski sign on the left disappeared, the right persisted.

Angiogram, May 23, 1946. It may be recalled that the clinical findings and pneumogram indicated that the lesion was on the left while the EEG showed it to

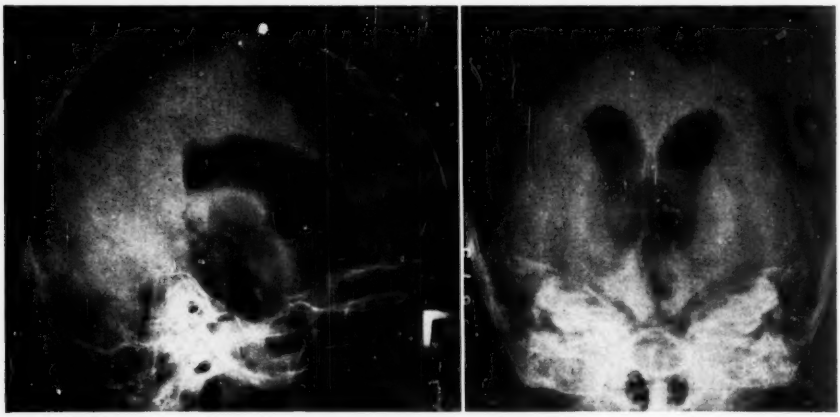

FIG. 6. Pneumogram shows evidence of cerebral damage beneath left anterior horn.

be on the right. On the left side the injection was made into the common carotid artery with the external carotid temporarily occluded. On the right side the external carotid was not occluded during the procedure. The radiopaque substance used was a 46 per cent solution of diodrast with a total dose of approximately 24 cc. for each side given in 2 amounts for stereoscopic purposes. During the injections the patient had momentary stimulation of respiration in spite of sedation. He was fortunately under rectal sodium pentothal anaesthesia, otherwise it is believed that he would have had a convulsion, which would be especially serious in this type of case. The angiogram on the right side demonstrated an aneurysm, about 1 cm. in diameter, communicating with the right anterior cerebral artery by means of at least two smaller branches (Fig. 7). The anterior cerebral arteries were stretched into a more open bow by the enlargement of the ventricles and the region of distribution was perhaps less well supplied with smaller branches. The aneurysmal sac retained the contrast medium (Fig. 8) for at least 7 sec. This aneurysm had apparently ruptured into the left frontal lobe and part one of the left lateral ventricle.

Operation. May 23, 1946. Right transfrontal craniotomy. The medial surfaces of the frontal lobes were adherent and yellowish fluid gushed up several times on elevat-

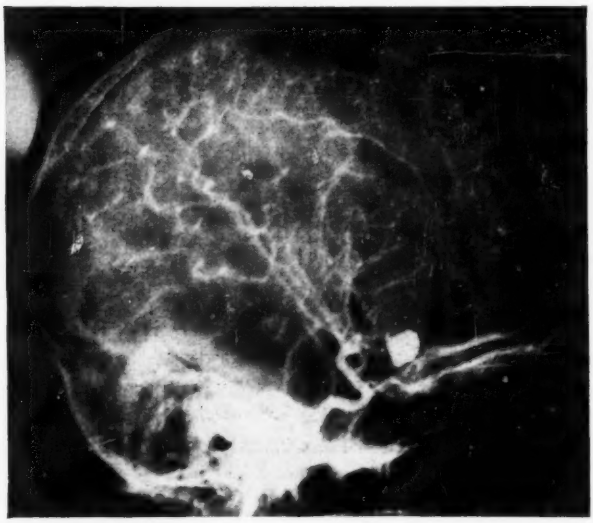

FIG. 7. First film of arteriogram in Case 2 with diodrast 46 per cent, showing aneurysm of right anterior cerebral artery. Note small vessels running to the aneurysm.

ing the right frontal pole. The ventricles were collapsed by ventricular puncture and by lumbar drainage of CSF. Following this the right frontal lobe was gradually separated from the falx and from the left frontal lobe, exposing at the base the rounded bluish aneurysm (Fig. 9).

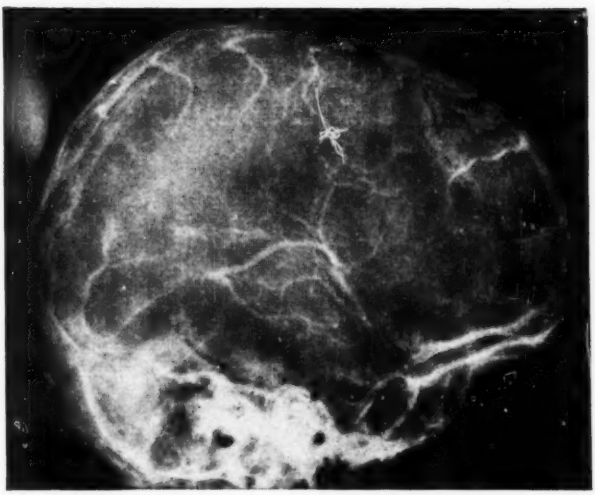

FIG. 8. Phlebogram film in Case 2, showing retention of diodrast in aneurysmal sac.

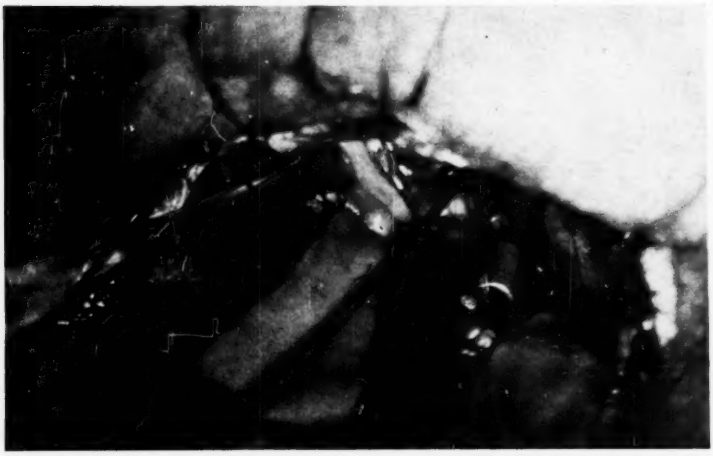

FIG. 9. The operative exposure, Case 2.

Four small feeder vessels were seen running to the sac as depicted in the angiogram, but only one of these appeared to come from the right side. They were divided in turn between tantalum clips, permitting the aneurysm to be lifted up gently, exposing a rather large vessel running beneath and medially. After these manipulations and when we were prepared to make the final delivery, the friable sac blew out unexpectedly with violent haemorrhage which was not satisfactorily controlled until three clips had been placed on a large vessel identified in a calmer moment as the right anterior cerebral artery. The sac was then excised and a dry operative field obtained before closure. The aneurysm had lain immediately above the optic chiasm. At the end of the procedure the optic nerve was seen to be undamaged. Postoperative x-rays show the position of the tantalum clips (Fig. 10.)

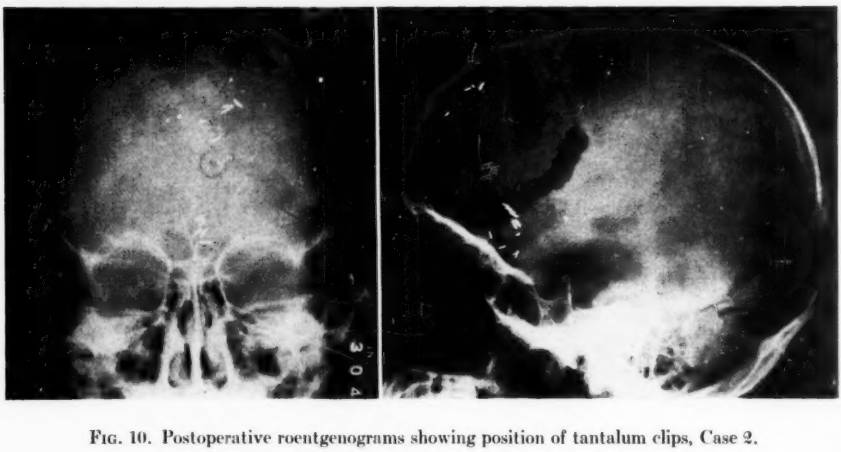

FIG. 10. Postoperative roentgenograms showing position of tantalum clips, Case 2.

Adhesions had prevented the safe placement of a loose ligature about the internal carotid artery within the skull and the ingenious method by Hamby[16] for occluding the carotid circulation in the neck from the cranial operative field was unsatisfactory in our hands for controlling the haemorrhage.

Pathological Note. The aneurysmal sac measured 1.5 by 1.2 cm. (Fig. 11A and B). The laminated wall varied from 0.5 to 3 mm. in thickness and was made up of friable, poorly organized thrombus (Fig. 11C). No elastic or muscle fibres were seen except in remnants of vessel wall stretched onto the outer surface of the sac. The only supporting tissue present was occasional collagen. The small arterial vessels penetrated the outer layer of the wall as noted at operation and in the angiogram.

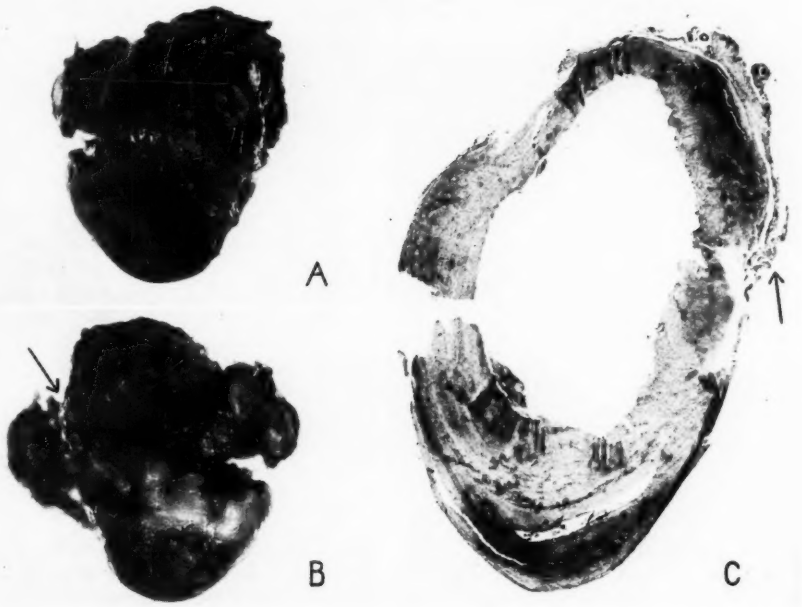

FIG. 11. Aneurysm removed in Case 2. (A) Inferior aspect (×2.5). (B) Superior aspect, showing small vessels, those on the medial side being indicated by arrow (×2.5). (C) Cross-section, showing irregularity of wall, loosely attached tissue in the cavity, and small vessels on the outer surface (×5.0).

This arrangement supports the theory of Padget[7] regarding the origin of congenital aneurysms from unresorbed embryonal vessels.

Postoperative Notes. For several weeks he showed lack of memory for recent daily happenings, but was bright and cheerful (Fig. 12). Incontinence cleared up when he began to get up and about the ward. In the EEG of June 20, 1946 (Fig. 5), 1 month after operation, there was an improvement of background activity but delta abnormality remained in considerable amount and was maximal in the right temporoparietal region. Lumbar puncture, June 19, 1946, showed clear fluid under normal pressure. On the day of discharge, June 28, neurological findings were essentially normal except for a slight left internal strabismus and an unsteadiness of gait apparently due to the rather long stay in bed.

Subsequent Course. The EEG of Aug. 5, 1946, 2½ months after operation (Fig. 5) continued to show generalized dysrhythmia with the greatest disturbance from the right hemisphere, with slow waves more localized to the frontotemporal region on the right but giving, on the whole, a record greatly improved as compared to that taken 1 month after operation. Neurological findings at this time were normal.

He showed progressive improvement and was in good physical health 11 months later. In the interval he returned to school and had been allowed to enter Grade 8 with his associates in September 1946, 4½ months after operation. His marks were generally poor except for passes in French and Science, and he failed his year.

He regularly defeated his parents at checkers and liked to play cards and chess. He was socially popular and happy but avoided athletics. Teachers were satisfied with his behaviour. When seen in 1947 the patient had continued to attend school.

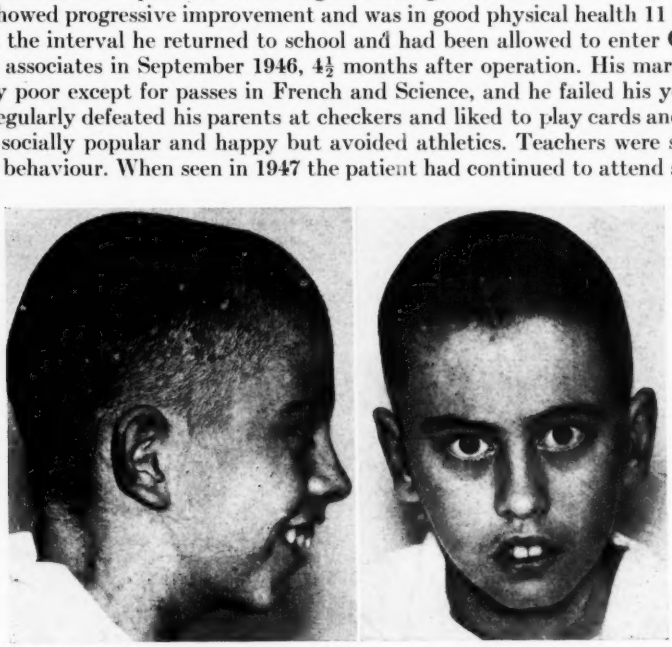

FIG. 12. Photograph of patient on 12th postoperative day.

His mental status was tested in some detail on several occasions by Dr. B. Silverman of the Mental Hygiene Institute. The boy's chief defect was poor memory and lack of initiative indicated by his failure to take interest in some former hobbies. He obtained poor results in tests designed to evaluate visual motor organization and new learning, but the results showed continual improvement, those of March 1947 being considerably better than those of September 1946.

The EEG of Oct. 25, 1947, showed a decrease of slow wave activity with an increase of rapid, more normal activity. Depression of alpha waves from the right occipital region as noted in a previous test was not seen. Residual general dysrhythmia was greater from right hemisphere, but with definite improvement.

When last heard from on June 29, 1949, it was stated that the patient had been generally backward at school. He had failed to pass from first year high school. He had been somewhat forgetful. On the other hand his general behaviour had been satisfactory and he was growing well. He had played baseball but cannot run fast. He rides a bicycle and goes swimming with other boys. He will probably be sent to a trade school. The EEG of July 6, 1949 suggested unresolved cerebral disturbance most marked in the right hemisphere.

DISCUSSION

Angiography. For vascular lesions of the brain especially we have preferred the method of surgical exposure, because of its reliability and safety. Moreover, ligations when necessary can be done simultaneously or through the same wound, and the radiopaque material can be directed to the external or internal division, or both at will.

There are no immediate untoward reactions to the injections of the carotid vessels with thorotrast, though one is naturally reluctant to use a radioactive substance. We never use more than 32 to 36 cc. in a selected case for bilateral injections, and generally never more than 24 cc. If the material should escape into the vascular sheath due to faulty injection, late scarring can be expected. With proper exposure and practice this should never occur.

Diodrast can be made as radiopaque as thorotrast but concentrations between 40 and 50 per cent must be used. We have used it and similar substances spasmodically since 1937. A concentration of approximately 45 per cent gave satisfactory visualization in Case 2 (Fig. 7). By percutaneous method we have been using 35 per cent diodrast which gives poor though adequate visualization but is still, though less, stimulating. Respiration may be accelerated and unless the patient is sedated twitchings or an epileptic seizure may result. Nevertheless it is a satisfaction to know that the material is rapidly excreted, so that repeat injections may be given if necessary. While rapid excretion of diodrast enables repeat injections to be made with some impunity, the potential danger of the medium cannot be discounted. It is felt that these iodine preparations may to some extent permeate through the vessel wall and to give rise to epileptic manifestations or in any event give rise to vascular spasm which may vary from case to case. One death is known to have occurred from the injection of this material and cases of hemiplegia following its injection are known to occur. Postmortem examination revealed a massive cerebral infarction. Why no organic chemist has linked the iodine atom to a more suitable molecule is difficult to understand.

Angiography makes possible an appropriately planned surgical attack on intracranial vascular lesions and in particular on saccular aneurysms. By this method the precise nature, localization, size and communications of the lesion may be determined. Pneumography, while of considerable value in the diagnostic work-up may, as in our Case 2 and in other published reports (Dandy,[7] Russel,[45]), actually indicate the wrong side by giving the localization of the retained haemorrhage, which may extend for some distance from the actual source of bleeding. Furthermore, as in Case 2, the clinical signs, such as ocular nerve palsy (Walsh and Love[53]), or even unilateral blindness (Jefferson[22]) may also point to the wrong side.

Electroencephalography. The EEG was correct as to the side of the lesion in both of our cases. The clinical picture and the electrographic disturbance are generally greater than one would expect from the amount of haemorrhage and brain damage disclosed at operation, and this suggests that the aneu-

rysm is capable of causing changes in the normal circulatory equilibrium with a resulting ischaemia of the cerebral tissue lying beyond the aneurysm.

Such effects of intracranial aneurysm on the intracerebral circulation require further study. Woodhall (mentioned by Dandy[7]) has previously noted 3 cases of aneurysm in which the side of the haemorrhage was indicated by EEG, and he assumed that the changes were due to a reduced blood supply to the brain.

Although the changes in the EEG record are not necessarily specific, they are similar in character and sequence to those described following experimental ischaemia due to the occlusion of the main arterial supply to the brain[50] and to disturbance of the cerebral circulation after simple exposure of the surface of the brain.[39]

In following the postoperative progress the EEG, a sensitive indicator of cerebral damage, continued to show disturbance at a stage when clinical examination revealed little abnormality, but when mental tests brought out deficiencies which were improving at a slower rate than the more gross clinical signs. The long period of recovery as indicated by the EEG is comparable to the results obtained in the study of head injuries (Jasper, Kershman and Elvidge[20,21]), where dysrhythmias may persist after neurological signs have vanished.

Surgical Treatment. The various methods that have been used for the treatment of intracranial aneurysms have been recently reviewed by Dandy,[7] Dott[9,10] and Jefferson.[24] In both cases reported here, tantalum clips[40] were used to isolate the aneurysm. In Case 1 this alone was sufficient to obliterate the aneurysm because of the small size of the sac and its disposition as a circumscribed dilatation of the arterial wall. Six clips were placed at strategic points. It seems unlikely that they could be forced open by the pulsation of blood. In Case 2 the aneurysm was excised *in toto*, and it is quite likely that, unless an attempt had been made to remove the sac completely, the final large vessel would not have been disclosed and would have remained as a source of recurrent bleeding.

The Use of Muscle. One of the earliest records of the use of muscle in the treatment of aneurysm is that of a case of a lesion of the internal carotid reported from Cushing's clinic by Sosman and Vogt,[48] where the sac was opened, packed with muscle and sewn up. The fact that muscle was employed successfully in 6 of the cases previously published indicates its value.

Nevertheless several unsuccessful experiences with muscle have been noted. Jefferson[22] mentions an instance in which muscle was packed about an aneurysm but recurrent fatal haemorrhage took place 18 months later. At autopsy the aneurysm appeared completely thrombosed except on its upper surface where it had ruptured. In 3 similar cases Jefferson[24] stated that 2 patients survived 4 and 5 years respectively, but in 1 of these the common carotid was tied 2 months later because of continuing pain which is "always a sign that the aneurysm is not securely thrombosed". In the 3rd, muscle was packed also around the aneurysm but fatal haemorrhage took place 1 month later.

This method, then, has not been entirely reliable and its value must depend among other things upon the structure and disposition of the aneurysm itself. The absence of muscle and elastic tissue from aneurysmal sacs is a common observation (Forbus,[13] Hermann and MacGregor[17]). Certainly in our Case 2 reconsideration of the histology of the aneurysm (Fig. 11) throws doubt on the value of bolstering a thrombotic sac with muscle. Moreover, the pressure of arterial blood coursing through the aneurysm must approach the limits of pressure (60 to 80 mm. Hg.) that freshly placed muscle clamps can resist (Horsley[19]).

Rupture of the Sac. Rupture of the aneurysm during operative manipulation is not uncommon, having occurred in 4 of the cases reviewed above, in our Case 2 and in instances of aneurysms located on other intracranial arteries.[14,33] It is therefore worthwhile to plan some means of dealing with this eventuality. This may be done most readily by carotid occlusion, either by ligation in the neck, preliminary to direct intracranial approach, or by clipping the artery intracranially, or by placing a loose ligature about the artery in the middle fossa before attempting to deal with the aneurysm. We have since noted that this latter method was used effectively in an early case reported from Cushing's clinic.[48] The authors prefer now to place a loose ligature round the artery in the neck which can be occluded by an assistant. As already stated, the ingenious method proposed by Hamby[16] for occluding the carotid circulation in the neck from the cranial operative field was attempted in our Case 2, but for technical reasons did not control the haemorrhage.

If permanent closure of the common or internal carotid artery is contemplated, some idea of the patient's tolerance to the procedure should be gained before operation. The percutaneous compression test of Matas has been found reliable by some[7] but has proved inconsistent in the experience of others.[24,36] The present authors prefer to ligate the vessels in two stages under local anaesthetic. Angiography according to some has been of value in determining to what degree the opposite carotid is capable of contributing to the blood supply of the hemisphere on the ligatured side. This can be done by compressing the common carotid on the side of the lesion while injecting the contrast medium into the carotid on the opposite side (Dott[10]), a technique similar to that used by Kristiansen[29] to demonstrate simultaneously both middle cerebral arteries for the diagnosis of temporal lobe vascular lesions. An additional test procedure has been the investigation of the effects of temporary carotid occlusion on EEG records, as described by Rogers,[42,43] by Chusid and Mahoney,[3] and in unpublished cases by ourselves. Although no case with serious complications after carotid ligation has as yet been available for EEG study,[44,54] this method appeared to be a most promising one, particularly when its results could be combined with the information derived from angiography.*

* In a recent case hemiplegia developed 10 hours after 2nd stage ligation of the common carotid (external had been ligated at the 1st stage with partial ligation of common). This was not predicted by EEG studies. A further observation has now been made of a relatively normal EEG recorded during the development of a hemiplegia from carotid ligation.

We have recorded brain waves during selective closure of the internal carotid system, but our experience is not yet sufficiently clear to indicate that it is a reliable test.

SUMMARY

1. The literature of the surgical treatment of anterior cerebral aneurysms is reviewed and 2 new cases are reported in which the aneurysm was demonstrated by stereoscopic angiography and treated by direct operative approach, in one case by trapping and clipping an aneurysm of the anterior communicating artery and in the other by clipping and excision of an aneurysm of the right anterior cerebral artery.

2. The value of angiography, not only in confirming the diagnosis of intracranial aneurysm, but also in revealing information upon which to base a planned surgical treatment of the lesion is re-emphasized.

3. EEG was of aid in the diagnosis and follow-up in both cases and indicated residual cerebral damage even after 3 years.

4. Both patients showed satisfactory recovery although clips were placed in the one case on the left, and in the other on the right anterior cerebral artery.

5. Both patients showed clinical pictures, the severity of which was out of proportion to the amount of haemorrhage or cerebral damage disclosed at operation, and it is suggested that this was partly due to the aneurysm producing a mechanical disturbance in the blood supply to the cerebral tissue.

6. Some advantages of excision of the aneurysm as opposed to other methods of local treatment are pointed out.

7. From a consideration of the histology of the aneurysm in our Case 2 and of reports in the literature it is suggested that the use of muscle to bolster up an aneurysmal sac may not be reliable.

8. It is pointed out that rupture of an intracranial aneurysm occurs so frequently during operative manipulation that some method of dealing with this eventuality should be planned beforehand. We suggest further trials of the method of placing round the internal carotid artery intracranially a loose ligature which can be used in emergency. A loose ligature round the carotid artery in the neck under the control of an assistant probably offers the safest method.

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STUDIES ON THE SACRAL REFLEX ARC IN PARAPLEGIA*

I. RESPONSE OF THE BLADDER TO SURGICAL ELIMINATION OF SACRAL NERVE IMPULSES BY RHIZOTOMY

ARNOLD M. MEIROWSKY, M.D., C. DAVID SCHEIBERT, M.D., AND THOMAS R. HINCHEY, M.D.†

Section of Neurological Surgery, Veterans Administration Medical Teaching Group, Kennedy Hospital, Memphis, Tennessee

(Received for publication May 19, 1949)

IT IS the purpose of these studies to broaden the clinical and experimental evidence of true bladder automaticity, and to apply this evidence to surgical measures in the treatment of patients with transverse myelopathy.

Goltz and Ewald[10] in 1896, and Müller[18] in 1902, apparently first observed automatic micturition subsequent to complete destruction of the sacral cord in animals and men. Upon sectioning of both pelvic nerves, Elliott[7] found temporary paralysis of micturition which was replaced 4 to 5 weeks later by some "lame" degree of bladder automaticity. Denny-Brown[5] described the occurrence of periodic micturition without residual and without incontinence in cases of complete destruction of the cauda equina. While Müller's and Denny-Brown's observations indicated the existence of automatic properties of the bladder, surgical denervation of the bladder in man to establish automaticity has never been reported. In 1945, Hoen[12] described 1 case in which he had sectioned the motor components of both 3rd sacral roots within the dural sac with the idea of lessening the tone of the external sphincter. Following this operation, the patient was able to evacuate his bladder completely. Hoen assumed that the 3rd root is the main contributor to the pudic nerve, but that contributions from the 2nd and 4th sacral roots were sufficient to maintain sphincter control. In a discussion of the surgical relief of spasticity in paraplegia, Freeman and Heimburger[9] suggested that section of selected anterior sacral roots may be indicated in some cases of neurogenic bladder that are resistant to routine methods of care. Recently, Heimburger, Freeman, and Wilde[11] succeeded in demonstrating accurate determinations of the sacral innervation of hypertonic bladders by novocain injections of the nerve roots in conjunction with cystometric studies. On the basis of their clinical investigation, they concluded that the anterior root of the 3rd sacral nerve rendered dominant innervation to the human bladder. They have furthermore shown that selected cases of neurogenic bladder, charac-

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† Present Address: Department of Neurological Surgery, Washington University School of Medicine, St. Louis, Missouri.

terized chiefly by urinary retention, can be treated successfully by bilateral block of the 3rd sacral roots with novocain solution or with alcohol. Shelden and Bors[19] reported that a voluntary type of micturition was established in 19 out of 24 patients subsequent to intrathecal injection of alcohol.

This paper deals entirely with observations on the bladder status in transverse myelopathy subsequent to surgical elimination of sacral nerve impulses by rhizotomy.

During the period from September 1947 until October 1948 12 rhizotomies have been performed for surgical relief of spasticity in the lower extremities, and 1 rhizotomy for relief of root pain and establishment of bladder automaticity. Two of these patients had traumatic transverse myelopathy of the cervical cord, while the remaining 11 patients had a thoracic level. The classical anterior rhizotomy with section of the anterior roots from T12 to S1 was performed on 5 of the 13 patients. This group of 5 patients is irrelevant to the present study of surgical elimination of sacral roots. It is, however, of interest to note that there was no change in the bladder status in these 5 patients subsequent to anterior rhizotomy.

Of the remaining 8 patients, all anterior roots from T12 to S5 were sectioned in 3 patients; anterior and posterior roots from T12 to S5 were divided in the other 5 patients.

REPRESENTATIVE CASES

Case 1. H.R., a 25-year-old white male veteran, sustained a gunshot wound of the left chest and thoracic spine July 1, 1945 on Okinawa. The injury resulted in immediate complete paraplegia with a dermatome level at T7. Progressive flexor and adductor spasm of the lower extremities was first noticed 2 weeks after injury. The patient was admitted to Kennedy Veterans Administration Hospital on June 6, 1946. Laminectomy of the 5th, 6th and 7th thoracic vertebrae (AMM) on June 13, 1947 verified a complete anatomical transection of the spinal cord at T5. Multiple decubital ulcers and progressive nutritional deficiency complicated the paraplegia-in-flexion. On April 15, 1948, anterior rhizotomy from T12 to S5 (AMM) resulted in complete flaccidity of the lower extremities.

Bladder status before and after rhizotomy (Table 1): Straight catheter drainage was used for 26 days subsequent to injury. A suprapubic cystostomy was performed on July 27, 1945 and was closed on April 26, 1946. After 1 month of straight catheter drainage, the patient was without a catheter for 8 months. During this time the patient had an overflow incontinence during spasm. In January 1947, a urethral catheter was reinserted with intermittent tidal and straight drainage. Seven days after anterior rhizotomy on April 15, 1948, the catheter was permanently removed. Since that time the patient has urinated at will without incontinence and without residual urine.

Comment. Following anterior rhizotomy the patient has an automatic bladder with voluntary micturition without residual and without incontinence.

Case 2. J.B., a 25-year-old white male veteran, was pinned under an auto when a jack broke on Feb. 26, 1947, with fracture of the 11th thoracic vertebra. He suffered

TABLE 1

Name	Date	Block	Sphct.	Pressure Cm/H_2O								Capacity	Void	Residual
				50 cc.	100 cc.	150 cc.	200 cc.	250 cc.	300 cc.	350 cc.	400 cc.			
H.R.	Apr. 12, 1948			14	22	28–70	30–70	38	55–70			360	240	120
	Apr. 15, 1948		Anterior Rhizotomy T12–S5											
	Apr. 20, 1948			7	10	12	16	20	26	36	50	480	480	6
	May 3, 1948			11	15	17	19	26	28	36	36	400	400	22
	May 10, 1948													2
	Jul. 28, 1948		31	3	4	9	12	14	16	20	22	425	425	0
	Sept. 10, 1948		74	7	15	17	22	30	34	37	40	400	400	0

immediate complete paraplegia with a dermatome level at T12. Progressive sharp radiating pain of L1 distribution in the left groin was present at the time of admission to Kennedy Veterans Administration Hospital on July 2, 1947. A thoracolumbar laminectomy of T11, T12 and L1 (AMM) on Oct. 30, 1947 revealed a yellowish discoloration with cystic degeneration of the cord at the level of the 11th thoracic vertebra with atrophy below. Novocain block of the left 1st lumbar nerve relieved the root pain. Automatic bladder function without residual urine or incontinence, as well as relief of pain, resulted under the influence of low spinal anesthesia. After anterior and posterior rhizotomy of T11 to S5 (AMM) on Sept. 1, 1948, the patient had relief of pain and automatic bladder function.

Bladder status before and after rhizotomy (Table 2): Straight urethral catheter drainage was used until July 1947, at which time patient was placed on tidal drainage for 6 weeks. He was then without a catheter with overflow incontinence due to bladder spasm until the laminectomy on Oct. 30, 1947. Straight catheter drainage was then utilized. Seven days after rhizotomy, the indwelling catheter was permanently removed. Since that time the patient has urinated at will, without residual urine and without incontinence.

TABLE 2

Name	Date	Block	Sphct.	Pressure Cm/H_2O								Capacity	Void	Residual
				50 cc.	100 cc.	150 cc.	200 cc.	250 cc.	300 cc.	350 cc.	400 cc.			
J.B.	Jul. 11, 1947	No		8	13	22	26	34				275	—	—
	Jul. 18, 1947	No		10	16	33	48			90		200	—	—
	Mar. 9, 1948	No	44–33	5	16	20	20	34	54			300	—	—
	Aug. 16, 1948	No	24	2–60	6–70	9–70	30	49				400	300	100
	Aug. 16, 1948	S2, S3, S4	18	5	6	8	18	30	40			400	400	5
	Aug. 20, 1948	No	31	0	0–60	4–70	6–70	10–70	13–70	20–70	21	375	275	45
	Aug. 26, 1948	Spinal	25	0	0	0	0	11	18			300	—	3
	Aug. 30, 1948		Anterior Posterior Rhizotomy D11–S5											
	Sept. 10, 1948		29	0	4	5	5	6	10	13	16	475	475	0
	Sept. 15, 1948													0
	Sept. 22, 1948													3
	Oct. 13, 1948													2

Comment. Bladder automaticity without residual and without incontinence, resulted after anterior and posterior rhizotomy.

RESULTS

Of 5 patients with anterior and posterior rhizotomy (T12–S5), 1 did not show any change in the functional status of his bladder. Bladder automaticity with occasional incontinence resulted in 1 patient who is, however, able to empty his bladder without residual. Perfect bladder automaticity developed in 3 patients who can initiate micturition at will and empty their bladder without residual and without incontinence.

Of 3 patients with anterior rhizotomy (T12–S5) such perfect bladder automaticity developed in only 1. One patient in whom bladder automaticity developed is showing a gradual decrease in residual urine. Poor cooperation on his part, however, prohibited permanent removal of the catheter at time of this report. It is of interest to note that pre- and postoperative cystometrograms revealed no substantial change. Sacral novocain block subsequent to anterior rhizotomy, however, produced a relaxed bladder with increased capacity. This suggests that bladder tone may be maintained by posterior sacral root influences. Because of an existing suprapubic cystostomy and because of premature discharge from the hospital, studies were not conclusive in this patient. This case also suggests the possible influence on bladder tone of the posterior sacral roots.

Of all 8 patients submitted to rhizotomy of one form or the other, but with inclusion of the sacral roots, bladder automaticity was established in 6. Five of them are now without catheter drainage and have a residual ranging between 0 and 40 cc. Although complicating factors must be considered in the remaining 2 cases, for the sake of analysis they should be regarded as operative failures.

DISCUSSION

As it has been shown by various observers[1,5,6,7,14] in cases of anatomical and physiological transection of the cord or conus medullaris, the human bladder retains its segmental innervation through the 2nd, 3rd, and 4th anterior sacral nerves. On the basis of this segmental innervation, the reflex bladder develops. Independent of the level of transection of cord or conus, there occurs in a large number of cases, as commonly observed, dysfunction of this reflex with resulting urinary retention. While the reasons for partial or complete inhibition of the segmental bladder reflex remain obscure, Hoen's[12] and Heimburger, Freeman and Wilde's[11] observations seem to suggest that inhibitory impulses are included in the sacral reflex arc. Because it has long been proven that the sympathetic nervous system does not influence bladder function,[2,3,4,8,13,15,16,17] elimination of the entire sacral reflex arc would then release the bladder from any possible stimulating or inhibiting impulses from higher centers.

The cases presented in this study show that surgical duplication of a transecting injury of the cauda equina, as produced by anterior and posterior rhizotomy of the lumbar and sacral roots, may result in bladder automaticity. Therefore, under certain circumstances, the bladder can learn to govern its own function. This self-government may be sufficiently perfect to result in

micturition at will, without residual, and without incontinence. Deficiency of varying degree in the execution of self-government exists for unexplained reasons in some instances. It may well prove to be due to chronic inflammatory changes in the bladder wall.

It seems, then, that surgical elimination of sacral nerve impulses is an essential factor in the production of bladder automaticity. There is some evidence that posterior sacral nerve impulses may sufficiently maintain bladder tone to interfere with the development of an automatic bladder.

From the evidence, reported by previous observers, as well as from these studies, it can be concluded that the bladder possesses a functional mechanism of its own. Even though the nature of this auto-mechanism remains obscure, the knowledge of its existence and its surgical production is of clinical importance. There are innumerous paraplegic patients with segmentally innervated, but completely inhibited, bladders. Their bladder status requires permanent catheter drainage with all its concomitant dangers. Under spinal or caudal anesthesia, the auto-mechanism of the bladder can be studied in each of these patients. While the rhizotomies reported in this paper have been performed primarily for the purpose of relief of spasticity, or, as in 1 case, for relief of pain, there is no reason why this operation could not be performed to establish bladder automaticity in cases of proven transections of the cord. In the presence of severe spasticity, necessitating anterior rhizotomy for relief of spasm, preoperative studies can readily be performed for simultaneous preparation of an automatic bladder by extending the rhizotomy to include the sacral roots. The operation is contraindicated whenever these preoperative studies, consisting of spinal or caudal anesthesia or sacral nerve block, are not conclusive.

CONCLUSION

Bladder function in complete paraplegia is of two types, reflex and automatic. Reflex control of urination occurs in the segmentally innervated bladder which maintains a reflex arc with the sacral cord. Automatic micturition may result after this reflex arc has been interrupted. The automatic bladder is characterized by adequate coordination between bladder and sphincter, resulting in micturition at will with any volume of urine, without appreciable residual, and usually without incontinence.

SUMMARY

1. The case histories of 5 paraplegic patients with anterior and posterior rhizotomy from T12-S5, and of 3 paraplegic patients with anterior rhizotomy from T12-S5, have been analyzed. The history of one representative case of each has been reported in detail. The bladder status before and after rhizotomy has been described and discussed.

2. Bladder automaticity developed in 6 out of 8 cases. Those 6 cases included 4 patients with anterior and posterior rhizotomy from T12-S5, and 2 patients with anterior rhizotomy from T12-S5.

3. The response of the bladder to surgical elimination of sacral nerve impulses by rhizotomy has been discussed. In selected cases of paraplegia with urinary retention, the clinical value of this procedure has been pointed out.

4. Bladder function in complete paraplegia has been divided into two types, reflex and automatic.

We are indebted to Dr. L. W. Freeman and to Dr. H. G. Schwartz for their advice and encouragement.

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STUDIES ON THE SACRAL REFLEX ARC IN PARAPLEGIA*

II. DIFFERENTIAL SACRAL NEUROTOMY. AN OPERATIVE METHOD

ARNOLD M. MEIROWSKY, M.D., C. DAVID SCHEIBERT, M.D., AND THOMAS R. HINCHEY, M.D.†

Section of Neurological Surgery, Veterans Administration Medical Teaching Group, Kennedy Hospital, Memphis, Tennessee

(Received for publication May 19, 1949)

In a preceding communication[2] we have discussed the establishment of bladder automaticity in paraplegia by rhizotomy with inclusion of the sacral roots. This operation is particularly indicated in patients with severe spasm. To establish bladder automaticity in patients without spasm, it seemed desirable to find a less extensive operative method.

Heimburger, Freeman, and Wilde's[1] and our own results‡ with differential sacral novocain blocks in a large number of patients with an inhibited reflex bladder lead us to believe that differential sacral neurotomy often results in bladder automaticity. It is the purpose of this communication to advance a method of differential sacral neurotomy and its indications with an illustration of this method by 1 case.

METHOD

1. *Preoperative Diagnostic Studies*

(a) Bilateral novocain block of 2nd sacral root, 3rd sacral root, 4th sacral root, 2nd and 3rd sacral roots, 3rd and 4th sacral roots, and of 2nd, 3rd and 4th sacral roots.

(b) Cystometric and sphincterometric data subsequent to each block with determination of bladder and sphincter tone, bladder capacity, residual urine, and possible incontinence.

2. *Indications and Contraindications*

Only those patients should be selected for sacral neurotomy who under the influence of a differential novocain block show increase in bladder capacity, decrease in bladder and sphincter tone, and ability to void automatically with residual urine less than 50 cc. The occasional occurrence of incontinence without residual, in our opinion, does not present a contraindication and is preferable to a retention catheter.

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† Present Address: Department of Neurological Surgery, Washington University School of Medicine, St. Louis, Missouri.

‡ Unpublished data.

3. *Operative Procedure*

Prior to operation, needles are placed in the sacral foramina (Fig. 1). Skin markings are applied with the needles in place and checked by x-ray to confirm the position of the individual sacral foramina. This has proven to be valuable in our hands because of existing anatomical variations.

A longitudinal skin incision is centered over the middle sacral crest. Superiorly, it extends to the level of the posterior superior iliac spine. The inferior boundary corresponds to a point 1 cm. below the sacral cornua. The incision is then carried through the posterior layer of the lumbodorsal fascia

FIG. 1. X-ray showing needles placed in 2nd and 4th sacral foramina.

to the middle sacral crest which is formed by the sacral spinous processes. The tendinous origin of the sacrospinalis muscle is sharply severed from the sacral spinous processes. By use of a chisel or a sharp periosteal elevator, the sacrospinalis and the multifidus are dissected subperiosteally so as to expose the dorsal surface of the sacrum. In doing so, the articular crest is encountered bilaterally. Between the latter and the lateral sacral crest, the 4 sacral foramina are found. It is therefore necessary to retract the paraspinal muscles to the lateral sacral crests. This can readily be accomplished by the use of self-retaining laminectomy retractors. The 2nd, 3rd, and 4th foramina are now exposed bilaterally (Figs. 2 and 3).

The dorsal surface of the posterior sacral foramina is covered by thin ligamentous tissue. In the fatty tissue beneath this ligament, numerous terminal branches of the lateral sacral artery are encountered. These branches must be cauterized. Upon pushing the cauterized vessels aside, the sacral nerves come into view. It is desirable to enlarge the sacral foramina slightly with a Kerrison punch. A small amount of bone is punched out medially and superiorly, but care is taken not to enter the dural sac. Enlargement

of the foramina facilitates hemostasis and identification of the nerves. As a rule, the sacral nerves are situated medially and quite superficially within

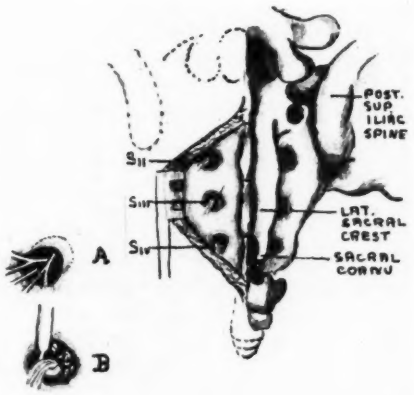

FIG. 2. Sacral neurotomy. Operative field is shown on the left. On the right soft tissues have been removed to indicate underlying bone structures. (A) Lateral sacral vessels emerging from sacral foramen. (B) Sacral nerve after cauterization of vessels and enlargement of sacral foramen. (Drawn by Dr. Anne D. Zinsser)

the posterior foramen. Occasionally, they may be found at a depth of as much as 1.5 cm. The nerve can easily be grasped with a blunt nerve hook, and must be pulled clearly into view to avoid possible injury to the rectal

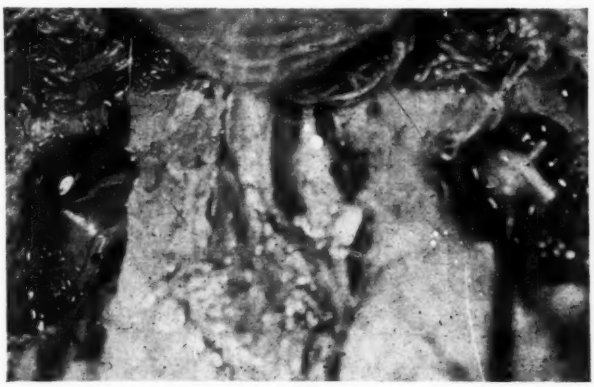

FIG. 3. Third sacral nerves lifted out of enlarged foramina.

wall. The nerve is then severed between two silver clips with a pair of fine dural scissors. Gelfoam is inserted into the sacral foramen. As many nerves are cut as has been determined preoperatively by novocain injections. Closure is performed in individual layers with interrupted sutures of medium and fine black silk.

CASE REPORT

C.H., a 27-year-old white male veteran patient, was stuck by a falling load of lumber on July 23, 1947, and received a compression fracture of the 1st lumbar vertebra. There was immediate complete motor and sensory loss below the level of the lesion. A laminectomy of the 1st and 2nd lumbar vertebrae was done elsewhere on July 24, 1947. One month later, the patient began to experience persistent burning and root-type pain of varying degree in the right lower thigh and knee. Two months after injury some improvement in sensory perception and muscular power was noticed.

The patient was admitted to Kennedy Hospital on Jan. 13, 1948. X-ray studies revealed an incomplete decompressive laminectomy as also evidenced by a CSF block. A secondary laminectomy was performed (AMM) on March 1, 1948, revealing

TABLE 1

Name	Date	Block	Sphct.	Pressure Cm/H_2O								Capacity	Void	Residual
				50 cc.	100 cc.	150 cc.	200 cc.	250 cc.	300 cc.	350 cc.	400 cc.			
C.H.	Feb. 18, 1948	No		5	6	9	15	19	30	32-70		350	250	100
	Mar. 23, 1948	No		5	10	11	15	20	24	32	38	400	0	400
	Apr. 17, 1948	No		23	25	29	30	39	47	55	65	550	0	550
	Apr. 19, 1948	S3		15	19	25	28	28				250	0	250
	Apr. 23, 1948	S2		15	26	32	36	36				480	0	480
	Apr. 28, 1948	Caudal		8	8.5	9	9	9	16	21	26	630	630	3
	May 2, 1948	S2, S4		10	10	14	12	25				635	210	425
	May 10, 1948	S3, S4		0	0	0	0	0	0	13		500	500	0
	May 24, 1948	S3, S4		0	0	0	0	0	0	7	7.5	470	470	
	Aug. 16, 1948		46	5	9	11	13	18	40			400	30	375
	Aug. 18, 1948	S3, S4	24	0	4	5	6	7	13	16	22	500	500	0
	Aug. 27, 1948		Sacral Neurotomy S_3, S_4 Bilateral											
	Aug. 28, 1948		25	0	0	0	0	2	4	8	10	460	450	10
	Sept. 2, 1948	—	—	—	—	—	—	—	—	—	—	—	—	15
	Sept. 15, 1948	—	—	—	—	—	—	—	—	—	—	—	—	0
	Sept. 30, 1948	—	—	—	—	—	—	—	—	—	—	—	—	8
	Oct. 12, 1948		54	8	9	12	16	18	19	—	—	—	—	5

compression of the conus medullaris at the lower level of the 12th thoracic vertebra. Subsequently, there was additional return of motor and sensory function.

The patient now walks with the aid of long leg braces with fairly good muscular function in both hips and the left thigh. Sensation is intact through the 3rd lumbar dermatome on the left and 2nd lumbar dermatome on the right. Normal sweating is present in the lower extremities.

Bladder Status Before and After Sacral Neurotomy on Aug. 27, 1948. From July 23, 1947, to Jan., 1948, an indwelling straight catheter drainage was used. Tidal drainage was employed until June, 1948, at which time a leave of absence was granted for 60 days with straight catheter drainage. Previous sacral injections of the 3rd and 4th sacral nerves with novocain revealed a relaxed bladder without residual urine (Table 1). The patient was unable to void without interruption of sacral nerve impulses.

On Aug. 18, 1948, the establishment of an automatic bladder was again confirmed by a sacral nerve block through the 3rd and 4th sacral foramina. A sacral neurotomy of the 3rd and 4th sacral nerves was performed on Aug. 27, 1948. The

following day the cystometric curve and sphincter tone coincided with the results of the preoperative novocain injections. Residual urine was 10 cc. The catheter was removed on the 10th postoperative day. Since that time the patient has voided at will without residual urine and without true or paradox incontinence. The patient has retained sensation of bladder fullness and can stop the urinary stream at will merely by cessation of straining.

DISCUSSION

With the intent of finding an operative method, technically easier and less extensive than the classical rhizotomy within the spinal canal, we have described a method of sacral neurotomy by which any number of anterior and posterior sacral nerves can be sectioned through the sacral foramina. In our indications for this operation we have been guided by preoperative sacral novocain blocks. While in some cases complete elimination of the segmental sacral innervation results in bladder automaticity, there are other cases in which only differential sacral neurotomy is required. The reasons for such variations remain obscure. The necessary extent of the segmental denervation can be determined by differential sacral novocain blocks, simulating temporarily a status of denervation. Our practical experience with this operative method is limited. While results with this surgical method are doubtful whenever the sacral blocks do not result in complete bladder evacuation at will, the reported case shows the effectiveness of the operation under optimum condition. Experience in a large number of cases must be gained before the reported surgical method can be standardized.

SUMMARY

1. The response of the bladder to surgical elimination of sacral nerve impulses in paraplegia has been reviewed.

2. An operative method for sacral neurotomy has been described.

3. Preoperative diagnostic studies, indications, and contraindications have been discussed.

4. Establishment of bladder automaticity by differential sacral neurotomy has been demonstrated in 1 case of paraplegia with complete urinary retention, secondary to physiological transection of the conus medullaris.

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MENINGIOMA OF THE OPTIC FORAMEN AS A CAUSE OF SLOWLY PROGRESSIVE BLINDNESS

REPORT OF 3 CASES

WINCHELL McK. CRAIG, M.D., AND LOUIS J. GOGELA, M.D.*
Section on Neurosurgery, Mayo Clinic, and Mayo Foundation, Rochester, Minnesota

(Received for publication May 31, 1949)

MENINGIOMAS arising from within the orbital cavity make up only a very small segment of intracranial meningiomas. Primary intra-orbital meningiomas have been removed surgically and verified pathologically in 17 cases at the Mayo Clinic.[1] Included in this group of intra-orbital meningiomas are those tumors that arise from the meninges within the confines of the optic foramen and that subsequently tend to extend both intra-orbitally and intracranially. These latter form an interesting subgroup in that, aside from their extreme rarity, they grow at an unusually slow rate and demonstrate a remarkable tendency toward bilaterality. The only symptom associated with the foraminal meningiomas is a slowly progressive blindness not accompanied by headache or pain.

The first reported instance of the "synchronous occurrence" of meningeal tumors involving both optic nerves was that described by Schott[5] in 1877 as an incidental finding on postmortem examination in the case of a 55-year-old blind charwoman who died of hepatic cirrhosis. The tumors were the size of a bean and were intimately attached to the arachnoid sheath of each optic nerve.

To our knowledge, the only other case of such bilateral tumors is that reported by Dandy[3] in 1922. The neoplasms were diagnosed clinically, and an intracranial operative procedure was used for the first time in the treatment of such lesions. Dandy particularly stressed the need for distinguishing these tumors from optic neuritis because he felt that unnecessary blindness could be prevented if the true situation were recognized sufficiently early. His case was that of a 13-year-old girl who had had progressive bilateral loss of vision for a period of 7 years. At operation he discovered collar-like growths that encircled the optic nerves and extended 0.75 cm. intracranially and 1.5 cm. intra-orbitally from their attachment at the point where the dura is "reflected" as it passes through the optic foramen. Dandy also reported the case of an 8-year-old boy who had noted bilateral loss of vision but in whom he found only a single neoplasm.

These foraminal meningiomas should not be confused with the small tumors arising along the mesial part of the sphenoid ridge and producing visual symptoms secondarily as a result of their encroachment upon the optic nerve.[4]

Meningiomas arising from within the optic foramen have been removed

* Fellow in Neurosurgery, Mayo Foundation, Rochester, Minnesota.

surgically in 3 cases at the Mayo Clinic. In 2 instances the tumors were bilateral and in the 3rd it was unilateral.

REPORT OF CASES

Case 1.—A 46-year-old white man noted the onset of progressive loss of vision in the left eye 10 years prior to examination. Within 3 years he was completely blind in that eye. Four years later vision in the right eye began failing, slowly and progressively.

Ophthalmologic examination disclosed that vision was reduced to 6/15 and was limited to the nasal field of the right eye. The right optic disc was pale and was elevated about 2 D. The left disc appeared even paler and the disc margins were blurred. Roentgen examination of the skull and the optic foramina gave normal results.

The visual loss was attributed to a chiasmal lesion, one that originally had been

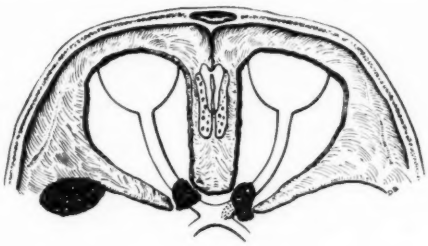


FIG. 1. (Case 1). Bilateral meningiomas attached to the optic sheath at the optic foramina and encircling the optic nerves. The tumor on the right was lobulated and measured 1 cm. in diameter. That on the left was smaller. A third meningioma was located along the left sphenoid ridge.

prechiasmal on the left. A left transfrontal craniotomy was carried out and 3 distinct tumors were encountered (Fig. 1). The first was situated along the outer third of the sphenoid ridge and measured 2.5 by 2.5 by 2.0 cm. It was removed without difficulty. When the optic chiasm was exposed, a small, cufflike, pinkish-purple mass was seen encircling the left optic nerve at the margin of the optic foramen. The entire tumor was removed. A similar, but slightly larger tumor measuring 1 cm. in diameter was found encircling the right optic nerve. One small lobule of this mass extended down into the sella and another to the right of the optic nerve. Only partial removal was possible because of its firm attachment to the optic nerve at the foramen.

The patient's impaired vision subsequently progressed gradually to complete blindness.

Case 2.—A 30-year-old housewife first became aware of failing vision in the left eye at the age of 12 years, and over a period of several years gradually became completely blind in that eye. The cause was never determined and the patient felt well otherwise. Eighteen months prior to examination, she noted the onset of progressive diminution of vision in the right eye. There were no other symptoms.

Physical findings were normal with the exception of those relating to the eyes. There was no proptosis and the eyes rotated normally. Vision was reduced to perception of moving objects in the temporal field of the right eye. Ophthalmoscopic examination disclosed marked pallor of the right disc with residual edema of 2 D. and similar extreme pallor of the left disc but with moderate loss of disc substance

temporally. Roentgen examination of the head and of the optic foramina gave normal results.

The lesion was suspected to be located in the region of the optic chiasm, and a right transfrontal craniotomy was performed. Upon elevation of the right frontal lobe, small, dual, cufflike tumors were encountered encircling and compressing the

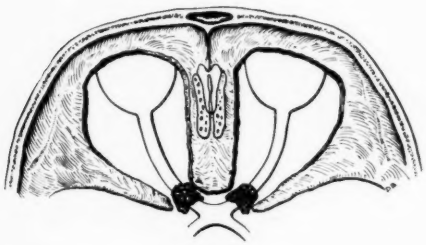


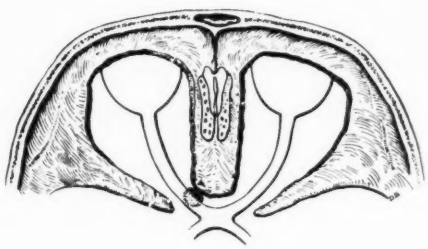
FIG. 2 (Case 2). Small, symmetric, bilateral, cufflike meningiomas arising within the optic foramina.

optic nerves at the optic foramina (Fig. 2). The tumors were dissected free from their attachments within the rims of the optic foramina with a blunt hook, and the dorsum of the optic canal on that side was decompressed.

The patient convalesced without incident and a few months after dismissal reported that she had noted some subjective improvement in the vision of her right eye.

Case 3.—A 41-year-old housewife experienced progressively diminishing visual acuity in the left eye over a period of 12 months. Examination revealed 6/20 vision in the right eye and 6/60 in the left eye. A large, dense cecocentral scotoma was

FIG. 3 (Case 3). A tiny meningioma located along the inferomedian surface of the optic nerve within the optic foramen and measuring not more than 4 mm. in diameter.



found in the field of the left eye. The left optic disc was pale. Roentgen examination of the head revealed early pressure erosion of the floor of the sella turcica. Pneumoencephalograms were normal and further observation was recommended.

When examined 10 months later, vision in the left eye was so impaired that movements of the hand were perceptible only at a distance of 2 m. and the pallor of the left optic disc had progressed. The progression of symptoms was suspected to be due to a small tumor impinging upon the left optic nerve. The optic chiasm was explored through a left transfrontal craniotomy. A small reddish, granular tumor was encountered lying along the inferomedian surface of the optic nerve within the optic foramen (Fig. 3). The proximal third of the orbital roof was removed and the

tumor was found to measure not more than 4 mm. in diameter. The atrophic optic nerve was sacrificed and the neoplasm was removed in its entirety with a curet.

The patient recovered without incident. On recent re-examination she was asymptomatic and the vision in the right eye had remained unchanged.

COMMENT

All the tumors encountered in these 3 cases were meningiomas of the psammomatous variety and all had exhibited an amazingly slow rate of growth. The presence of meningiomas attached to the sheath of the optic nerve may be explained adequately by the clusters or "caps" of arachnoidal cells occurring so abundantly along the intra-orbital course of the optic nerve as suggested by previous investigators.[2] In all probability, the cufflike growths arising within the optic foramen are but "sheath" meningiomas situated within a specific location. However, it is difficult to explain why this special predilection should exist and why these growths should demonstrate a tendency toward bilaterality. Microscopic examination of normal optic nerves and their sheaths (Fig. 4) does not disclose the clumping of arachnoidal cells to be any more frequent or more exaggerated within the optic foramina than anywhere else along the intra-orbital course of the optic nerve.[1] Yet, 2 of the 3 patients presented bilateral tumors, 1 of the growths appearing fully 16 years after its mate.

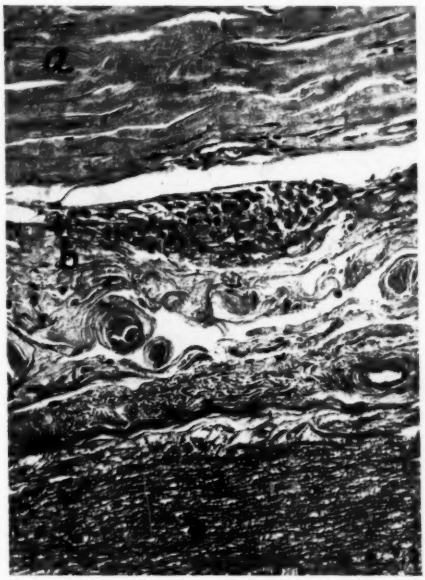


FIG. 4. Normal optic nerve and sheath (hematoxylin and eosin ×200). *a*, Dura; *b*, cluster of arachnoid cells from which meningeal tumors supposedly arise; and *c*, optic nerve.

Review of these cases serves to emphasize the fact that slowly progressive blindness, unaccompanied by headache or pain, is the only symptom to be expected in the presence of these tumors. Unfortunately, in all 3 instances blindness had been allowed to advance to the point at which damage to the optic nerves had become irreparable before the probable nature of the lesion was suspected. Surgical intervention in all these cases served only to establish the diagnosis and probably had no therapeutic effect. There is every reason to believe that with early recognition of these lesions and their prompt removal, failing vision may be controlled and possibly even improved or

restored. With the aid of the ophthalmologist and the technics that he has at his disposal, including carefully performed studies of the visual fields, it is possible to distinguish these tumors from non-surgical lesions of the optic nerve. The possible presence of a surgical lesion such as a foraminal meningioma should be suspected in all instances in which the patient presents himself with progressive, painless loss of vision due to a lesion of the optic nerve.

SUMMARY

Meningiomas arising from within the confines of the optic foramen are extremely rare and are of interest in that they tend to occur bilaterally and grow at an amazingly slow rate. Three additional cases are reported in which meningiomas of the optic foramen were removed surgically. In 2 instances the tumors were bilateral.

Slowly progressive blindness, unaccompanied by headache or pain, is the only symptom presented with these lesions. Early recognition and prompt surgical removal is paramount in the treatment of such tumors.

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FOUR CONGENITAL TUMORS FOUND AT OPERATION WITHIN THE VERTEBRAL CANAL

WITH OBSERVATIONS ON THEIR INCIDENCE

SAMUEL P. W. BLACK, M.D.,* AND WILLIAM J. GERMAN, M.D.
Department of Surgery, Yale University School of Medicine, New Haven, Connecticut

(Received for publication June 1, 1949)

THE epidermoid, dermoid, teratoid, and teratomatous tumors of the central nervous system are congenital neoplasms that are of particular surgical, pathological, and embryological interest. They result from a developmental defect of embryonic life and may occur intracranially or intraspinally. While not frequently encountered in either location, their presence should be considered, especially in a child, when evidence exists of a space-occupying lesion within the cranium or the vertebral canal. Numerous observations[1,2,3,4,5,8,10,15,16,17,18,21,25,30,31] have been made concerning their origin, incidence, structure, clinical aspects, and surgical treatment, but many details of their life histories remain to be clarified.

The discovery at operation of an intraspinal epidermoid and teratoid on successive days prompted a review, with special reference to similar cases, of the neuropathological material recorded at The New Haven Hospital during the past 20 years. In the course of that review an intraspinal teratoid and a teratoma were found. We wish to present those 4 cases and to comment briefly upon their incidence in relation to the series from which they were collected.

INCIDENCE

Since 1927 there have been 124 intraspinal tumors verified at operation in this hospital (Table 1). Included were those tumors that arose primarily

TABLE 1

Verified Intraspinal Tumors, The New Haven Hospital, 1927–1948

Meningiomas	24	Teratoids	2
Metastatic Lesions	21	Epidermoid	1
Neurofibromas	19	Hemangioblastoma	1
Sarcomas	16	Medulloblastoma	1
Vascular Malformations*	9	Spongioblastoma	1
Granulomas	7	Teratoma	1
Lipomas	4	Unclassified	8
Astrocytomas	3		
Chordomas	3	Total	124
Ependymomas	3		

* Diagnosed by inspection.

* George H. Knight Memorial Fellow, Laboratory of Physiology, Yale University School of Medicine.

from intraspinal structures and neoplasms that invaded the canal by metastasis or direct extension from primary sites elsewhere. With the exception of 9 hemangiomas which were encountered but not biopsied, all diagnoses were made on the basis of histological examination of tissue obtained at operation.

The epidermoid, 2 teratoids, and teratoma that were found comprise roughly 3 per cent of the intraspinal tumors. This figure may be unduly high because the series was compiled relatively shortly after the verification of 3 of the 4 cases. However, in view of the increasing numbers of these tumors that are being reported, they are apparently not as rare as was once supposed. Not included here are a sacrococcygeal teratoma nor two teratomatous cysts which were associated in one instance with a meningocele and in the other with a myelomeningocele.

During this same period there have been approximately 525 intracranial tumors verified histologically at operation. The criteria and classification of Cushing[11] were used for acceptance in the intracranial series. Four epidermoids and 1 dermoid were found, comprising about 1 per cent of the total number.

Of the whole series of approximately 650 cases, the epidermoids, dermoids, teratoids, and teratoma accounted for about 1.4 per cent. The larger number of intracranial epidermoids is worthy of note as was the absence in this series of teratoids and teratomas from that location.

CLASSIFICATION

There is not a uniformly accepted classification of these tumors. It is necessary, therefore, to state the histological structures defined by the terms epidermoid, dermoid, teratoid, and teratoma.

The epidermoids, the intracranial examples of which were beautifully described in a series of articles from Cushing's clinic[1,2,10,15] are composed strictly of epidermal tissue and the debris associated with it. They are of ectodermal origin and have been referred to as "tumeur perlée" by Cruveilhier,[9] "cholesteatoma" by Müller,[27] "Perlgeschwülste" by Virchow,[32] "epidermoide en piale" by Bostroem,[4] "nonhair-containing cholesteatoma" by Horrax,[15] and "pearly tumor" by Bailey.[1,2]

The dermoids, also derived from the embryonic ectoderm, differ from the epidermoids in that epidermal derivatives such as hair follicles, hair, or sweat glands are present in addition to the epidermis. They have generally been recognized as a separate class of tumors. Horrax[15] suggested the name "hair-containing cholesteatoma" be applied to them. Bostroem[4] believed that the epidermoids and dermoids originated from embryonal ectodermal inclusions at the time the neuro-ectoderm was splitting off from the ectoderm that forms the skin. It is a theory that has important clinical application in view of the skin dimples and dermal sinuses that may be associated with them.[7,33] The intraspinal dermoids and epidermoids have recently been reviewed by Sachs and Horrax,[31] who found 59 previously mentioned in the literature and added 2 more cases.

As used here, the term teratoid designates a bidermal tumor, one in which may be recognized derivatives of the embryonic ectoderm and mesoderm. The classification of these tumors will benefit greatly from a clarification of their morphogenesis because under the heading of teratoid there may presently be grouped bidermal tumors ranging in complexity from a small body of fat associated with ectodermal derivatives, as in Case 2 of the present series, to a histologically complex mass resembling a teratoma but in which no endodermal derivatives can be identified.[5,17]

The teratomas, as indicated above, are composed of derivatives of all three germ layers. The recognition of those derivatives is not always easy because, as was stated by Hosoi,[17] "the ectodermal and mesodermal elements tend to overgrow at the expense of the endodermal elements, which may either become retarded in their growth or disappear entirely." A discussion of the origin of the teratomas has been nicely presented by MacCallum.[23] Fifteen intraspinal teratoids and 10 teratomas have recently been accumulated from a review of the literature by Sachs and Horrax.[31]

Unless otherwise stated, the cellular constituents of these tumors are benign, and the growths assert their presence by pressure on surrounding nervous structures. The possibility of malignant change in the teratoids and teratomas should be borne in mind, however, and act as an added incentive to early and complete removal.[18,23]

CASE REPORTS

Case 1. (N.H.H. B97158) N.R.T., a girl aged 8 and $\frac{9}{12}$ years, was admitted in May, 1947 with the chief complaint of pain in the back for 4 years. At birth she was noted to have a clubfoot bilaterally and a dimple in the midthoracic region of the back. There had been no discharge from the dimple and no abnormal hair grew about it. At 2 years there was noted an abnormal curvature of the spine to the left in the thoracic region, and it continued to curve increasingly to the time of admission. At 4 years the patient experienced the onset of attacks of sharp, brief, interscapular pain, occurring at first every 6 months but becoming a daily happening about 8 weeks before admission. With the increased occurrence of the pain, it was noted that her head was carried in such a manner that the chin was in the jugular notch, apparently in an attempt to straighten the normal cervical curvature. There was no sudden increase in growth prior to this. A generalized headache developed shortly before admission, and she moved about very cautiously. The pregnancy had been uneventful; there was no history of familial defects, and a younger sister is apparently normal.

Examination. The head measured $54\frac{1}{2}$ cm. in the largest circumference. Her chin was held in the jugular notch, and all motions of the neck were extremely limited in range. In the midthoracic region of the back a 2 mm. skin dimple was noted to the right of the spinal column. Elsewhere about the body the skin was normal except for an irregular, light brown, 2 cm. area beneath the right ear. The right leg was shorter and slightly smaller than the left. A "winging" of the scapula was noted when pushing on the left arm. There was an area of hyperesthesia over the back in the midthoracic region with the most sensitive portion about the skin dimple. The abdominal reflexes were bilaterally absent as were the knee and ankle jerks on the right. There was a positive Babinski response on the right.

Roentgenograms revealed narrowing of the anteroposterior diameters and widening laterally of the bodies of the 6th, 7th, 8th, 9th and 10th thoracic vertebrae with an increased interpedicular space. The spinous processes in this same region appeared bifid and the bodies of these vertebrae showed incomplete fusion with marked narrowing of the disc spaces. There was also a spina bifida involving the 3rd, 4th, and 5th lumbar vertebrae. A myelogram revealed almost complete obstruction at the level of T9.

Operation. The incision followed the curvature of the spine to the left and did not involve the skin dimple. The muscles were cleared from the lateral aspects of the bifid spinous processes from T6 to T10 and from the intact processes of T5 and T11. The cup formed by the bifid processes held a mass of fibrous and muscle tissue in which could be identified a thin fibrous structure running from the subcutaneous tissues underlying the skin dimple into a plate of bone which was found to connect the bases of the bifid spines of T9. A similar plate of bone was found to connect the bifid processes of T6, T7, T8 and T10. On entering the canal, the fibrous structure was seen to be attached to the dura, in which no pulsation was noted below T9. There was no evidence of extradural infection. The spinal canal was widened laterally in the region of the exposure. Upon opening the dura a smooth, round, white, 1.5 cm. mass was observed at T9 lying between the spinal cord which was divided into two equal halves about it. The tumor was covered with arachnoid and running into its superior portion was the fibrous stalk from the dura. Issuing from its inferior aspect were two small, white, strands of tissue which immediately united and proceeded caudally, not joining the cord in the region of the exposure. These strands were secured in a silver clip at their point of emergence from the tumor and cut through. It was then possible to lift the tumor and fibrous stalk from the spinal canal. The spinal cord was seen to bifurcate at the upper end of the incision at T5 and came together again at about T11. The tumor appeared to have slightly compressed the right half of the cord. No other abnormalities were noted within the region of the exposure and extra spinal roots were not identified. Upon removing the tumor the cord moved noticeably cephalad.

The *postoperative course* was very satisfactory, and she was discharged on the 10th day after operation. She was able to move her neck freely without precipitating the former pain; the Babinski responses were negative, and the abdominal, knee and ankle reflexes were equal and active bilaterally. She is at present going to school and leading a normal life.

Pathology. The specimen consisted of a spherical, cystic, white tumor, 1.5 cm. in diameter, to which was attached a thin, tough, fibrous piece of tissue. The lumen of the cyst was filled with a white, soft, material. Microscopically, the cyst wall was composed of squamous epithelium and the lumen was filled with laminated keratinized material and cholesterol crystals (Fig. 1). Diagnosis: Epidermoid cyst.

Comment. The structural abnormalities presented by this little girl appear to be morphogenetically related. The primary aberration would appear to be the inclusion of surface ectoderm in the neuro-ectoderm when the two separated during the first month of intra-uterine life.[4] The dimple and sinus remain as testimony to that event.[33] The included portion of surface ectoderm separated the neural plates so that the spinal cord was subsequently divided into two halves about it. In the formation of the vertebral column by the mesenchyme there resulted, because of the obstruction to an

orderly investment offered by the ectodermal sinus, the partial formation of two vertebral columns as displayed by the widened canal and duplicated spinous processes. There was a single dura and arachnoid, but a separate pia invested each half of the cord.

The sequence of clinical events possibly began by the tumor pressing against the right side of the cord, affecting the pyramidal tracts, and leading to underdevelopment of the right leg and consequent scoliosis. Once present the scoliosis accentuated the pressure of the cord against the tumor. This could account for the signs of pyramidal tract damage that the patient displayed. That the tumor, lying as it was in a bifurcation of the cord, was attached to the dura and to the vertebral column and skin by the fibrous

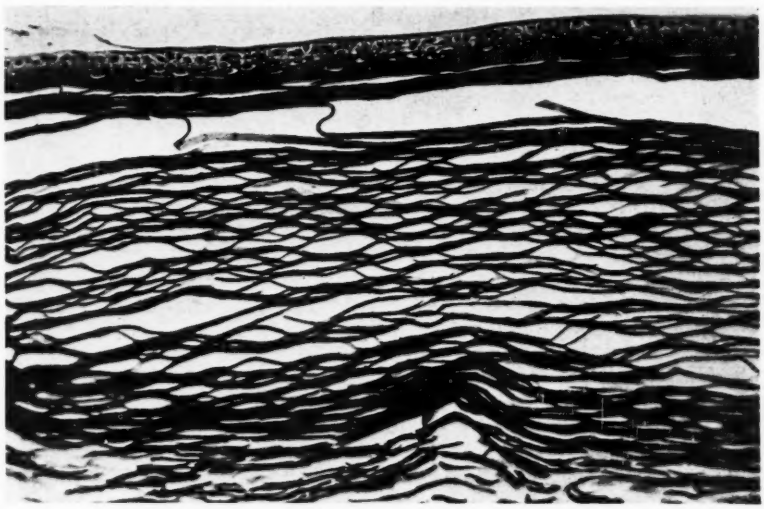

FIG. 1. Case 1. Photomicrograph showing stratified squamous epithelium lining cyst wall, and desquamated, keratinized material within lumen of the cyst. (Hematoxylin and eosin stain, ×230)

cord, contributes another interesting aspect. As growth of the axial skeleton proceeded, it is conceivable that some of this downward growth, in relation to the relatively fixed position of the intraspinal structures, was transmitted to the dura and cord by the tumor and its fibrous sinus tract. The contents of the posterior fossa were thus put under traction. The reversal of the cervical curvature resulted from an effort to have the shortest possible distance separating the site of traction and the posterior fossa. Although it was not possible to verify this theory,[29] indication of its correctness was afforded by the observation that after the tumor was removed the cord moved noticeably cephalad. Following the procedure the patient's headaches were completely relieved and the cervical curvature returned to normal.

In 1940 Herren and Edwards[14] reported on 43 cases of diplomyelia, also

known as diastematomyelia, double spinal cord, and duplication of the spinal cord, in only 3 of which was the diagnosis made during life. In none of their cases was a tumor situated between the divided halves of the cord. Such a finding was reported, however, by Harriehausen.[13] In his case a 5×2.5 cm. cylindrical tumor, one that would here be classified as a dermoid, was found at autopsy to lie between the halves of the spinal cord in the upper lumbar region. A skin dimple was not associated with it and the overlying laminae were normal. Since the report of Herren and Edwards, Lichtenstein[22] has recorded finding an example of diastematomyelia at autopsy, and Marr and Uihlein[24] and Maxwell and Bucy[26] have added cases in which the diagnosis was made during life. In the latter case a dimple was present in the lumbar region, and a dermal sinus led to and was attached at the point where the dura divided to enclose separately each half of the cord. Three additional cases were tabulated, but not elaborated upon, by Ingraham and Scott.[19]

Case 2. (N.H.H. B96979) T.W., a 20-year-old Yale undergraduate, referred by Dr. William Perham of New Haven, was admitted in May, 1947 with the complaint of pain in the lower back. Four months prior to admission he fell landing on the buttocks. There was a dull ache in the gluteal region for 2 hours but no further difficulty until 6 weeks before admission when he experienced a pain in the lower spine. It became more severe and radiated through the posterior aspect of the left thigh to the knee. The pain was made worse by coughing, straining, and moving about, and it often woke him at night. There were no other motor or sensory symptoms.

Examination. Slight flattening of the lumbar curvature and spasm of the erector spinae muscles bilaterally were noted. Flexion, extension, and rotation of the spine were limited, and all voluntary movements were performed slowly and guardedly. Percussion of the spine elicited pain beginning at T12 and increasing caudally to the middle of the sacrum. Straight leg raising was limited to 60° on the left and to 30° on the right. The knee and ankle jerks were equal and active bilaterally. No atrophy was noted in the extremities. There was diminished appreciation of pain in an area including the 4th and 5th lumbar and 1st sacral dermatomes on the left.

Roentgenograms of the spine were normal. Additional information was sought from myelography. The needle entered the canal at the 4th lumbar interspace, and the dura was pierced. The first few drops of fluid were grossly cloudy after which it became clear. The needle was then rotated in its long axis and withdrawn slightly. With this maneuver the fluid again became grossly cloudy. The needle was then withdrawn completely. No cells or organisms were seen in the fluid and cultures remained sterile. Following this he felt somewhat improved, but there was no change in the neurological findings.

Operation. There was no evidence of extradural pathology. At the level of the 4th lumbar vertebra the dura was slightly prominent, firm, and of a yellow color. Upon opening the dura the arachnoid was found to be adherent to it. Between the roots of the cauda equina and presenting posteriorly was a mass measuring about 2 cm. in diameter. Posteriorly it was composed of yellow adipose tissue while anteriorly it was cystic and continuous at its upper and lower poles with the filum terminale. At the lower portion of the cystic mass was a depressed lobule which appeared to have been previously tapped by a needle. The entire mass was dissected free of the roots of the cauda equina and removed.

Course. Due to an inability to void spontaneously, an indwelling catheter was necessary from the 2nd to the 4th postoperative day. The urine remained sterile. He was discharged on the 8th day after operation, relieved of his symptoms, and with full range of motion in his trunk and legs. The only sequela is a hypalgesia of the 5th sacral dermatome bilaterally.

Pathology. The specimen, which was attached to the filum terminale, measured 2.5×1.5 cm. It was composed of a mass of yellow adipose tissue, intimately attached to and partly surrounding a white, cystic, lobulated structure. Microscopically, the cystic portion was lined by squamous epithelium, and in its lumen was a small amount of keratinized epithelium. In the fatty portion adjacent to the cyst could be identified a considerable amount of loose areolar tissue, some sweat glands, and blood vessels (Fig. 2). Diagnosis: Teratoid of the filum terminale.

FIG. 2. Case 2. Photomicrograph showing cyst lined by stratified squamous epithelium; sweat glands, connective tissue and fat are also present. (Hematoxylin and eosin stain, ×52)

Comment. Myelography was attempted in this patient because the neurological findings were felt to be not entirely compatible with the tentative diagnosis of ruptured intervertebral disc. In retrospect, the approximate nature of the lesion was suggested by the sterile, cloudy fluid obtained at lumbar puncture. It was not until Case 1 was operated upon, however, that the significance of this finding was fully appreciated.

That the tumor proved to be teratoid was unusual. Kernohan and Fletcher-Kernohan[20] estimated that more than 50 per cent of the tumors of the filum terminale were ependymomas. Dermoids and epidermoids have been reported as either originating from or attached to it.[3,28]

Case 3. (N.H.H. B99551) J.DeF., a 10-month-old child, referred by Dr. John Buckley of Bridgeport, Connecticut, was admitted in July, 1947 with the complaint

of occasional "tremor" of the right foot. His postpartum development was otherwise normal. Early in pregnancy, his mother had had an attack of "intestinal flu," marked by malaise, headache, nausea, and diarrhea. She felt "very warm," but the temperature elevation, if any, was not recorded. She did not consider herself sick enough to call the family physician, and at the end of a week felt well enough to be up and about. The onset of the illness occurred 4 weeks after the last menstrual period prior to the birth of the patient. Three older siblings have developed normally.

Examination. The child appeared well developed except for the lower extremities. A "pilonidal dimple" was in the sacral region, and over the 2nd and 3rd lumbar vertebrae there was some telangiectasis, with an overgrowth of hair. There was a poorly sustained clonus in the right foot; a few spontaneous motions were noted in the left foot. He reacted to painful stimuli applied to both legs. The knee and ankle jerks were not obtained on either side, and the Babinski response was positive bilaterally.

Roentgenograms revealed an increased interpedicular distance in all the lumbar vertebrae and in the last thoracic and 1st sacral. There was also evidence of erosion of the posterior aspects of the vertebral bodies from T12 to L4. There was no defect in the vertebral arches.

Operation. The spinous processes of L1, L2, and L3 were small, and the laminae of the lumbar vertebrae were very thin. There was an absence of extradural fat in the lumbar canal. Upon opening the dura in this region a yellow, firm, 6×3 cm. mass covered by arachnoid bulged up into the incision. It was dissected free of the cord, conus, and upper portion of the cauda equina and completely removed. The tumor had markedly compressed the underlying nervous structures.

Course. The child recovered rapidly, although catherization was necessary for 3 days. On discharge 16 days after operation, a slight amount of active flexion in the thighs was noted. Twenty months later there was active flexion of the thighs, and he was able to bear his weight in the standing position. No sensory loss was demonstrable. The ankle jerks were briskly active and the Babinski responses were negative.

Pathology. The mass measured 6×3×2.5 cm. It was of firm, fatty consistency, rather rough to touch, and irregular in outline. Upon cutting into the mass, a cyst was encountered which was filled with white, soft material. Microscopically, the cyst was lined by squamous epithelium surrounded by collagenous tissue. The tissue adjacent to the cyst was composed mainly of fat in which could be identified sweat glands, nerve cells, nerve fibers, and striated muscle (Fig. 3). Diagnosis: Teratoid.

Comment. An interesting and possibly important aspect of the patient's history was the illness the mother had had in early gestation. Campbell[6] has mentioned the occurrence of a few cases of major neural axial deformity in patients with a family history that was negative for congenital defects, but in which was substantiated the fact that the mother had had an acute upper respiratory infection with associated hyperthermia to 104–105° F. during the 3rd and 4th week of gestation. The information obtained in our Case 3 is too poorly documented to offer it as another example of Campbell's observation. The similarity of certain aspects, however, in particular that dealing with the period of gestation during which the mother was ill, is thought worthy of comment.

*Case 4.** (N.H.H. A90789) E.L., a 5-month-old baby, was admitted in 1938 with the complaint of an increasing flaccid paralysis of the legs for 3 months. The pregnancy was uncomplicated and two siblings were living and well.

Examination. The lower extremities were flaccid, and motion in the arms was quite weak. Flexion, extension, and lateral bending of the head were resisted. The entire spinal column was tender to light percussion. She reacted to pinprick more vigorously when stimulus was applied above the 4th cervical dermatome. Reflexes were sluggish or absent. Repeated attempts at lumbar and cisternal puncture returned at most a single drop of clear fluid.

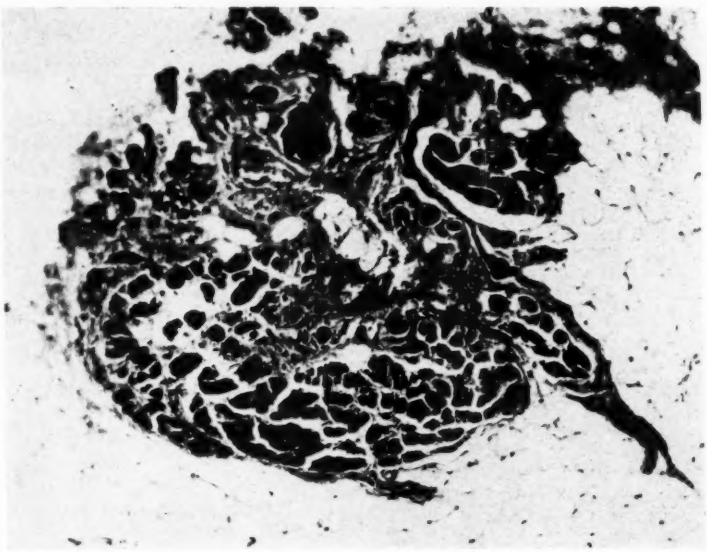

FIG. 3. Case 3. Photomicrograph showing darkly staining bundles of muscle and lighter staining connective tissue with surrounding adipose tissue. (Masson stain, ×130)

Roentgenograms revealed a widening of the entire canal, scoliosis of the thoracic and lumbar spine, and osteoporosis involving the long bones.

Operations. The patient underwent 3 operations which were carried out over a period of 5 weeks. They were all performed under local anesthesia, and she withstood them remarkably well. *1st Op.* Laminectomy was done from C4 to T2. The spinal canal was widened, and the dura was closely applied about it. Upon opening the dura and arachnoid a dark red mass, covered by pia but extramedullary, was found to occupy almost the entire canal. The upper end of the tumor, which lay at or just below the foramen magnum, extruded itself when the child cried during the procedure. The mass was gently elevated as far caudally as the incision permitted and there amputated. The greatly attenuated horseshoe-shaped cord was then seen

* Previously reported by German.[12]

lying anteriorly. *2nd Op.* The laminectomy was extended to L1. Throughout the greater portion of this exposure the tumor was buried within the cord. It was necessary, therefore, to split the cord between the posterior columns, following which it was possible to remove the tumor from its bed in a single piece as far as the incision would allow. *3rd Op.* The incision was extended to S2. In this region a skin-covered mass containing hair and caseous material was encountered. The lower extremity of the tumor lay at S2 while at the upper end a 1.5 cm. strand of it extended into the

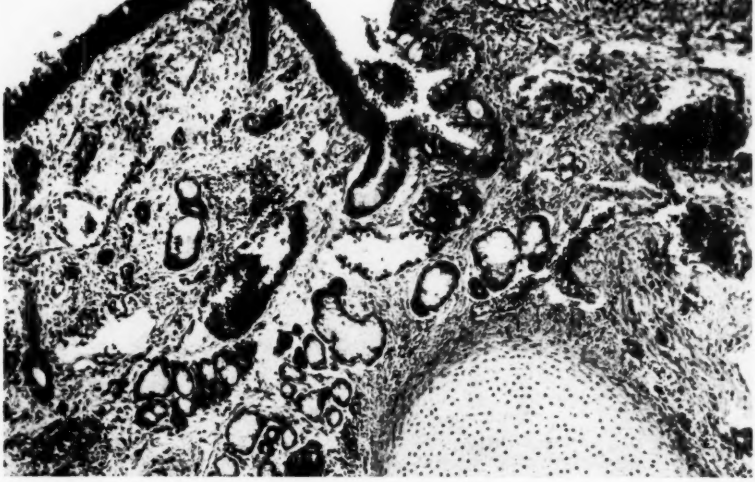

FIG. 4. Case 4. Photomicrograph showing cartilage, epithelial glands, and respiratory tract mucosa. (Hematoxylin and eosin stain, ×150)

conus medullaris. It was removed as completely as possible. Viewed over all, the tumor had apparently arisen from some intramedullary site, extruded itself through the posterior aspect of the cord and extended up and down the canal.

Course. Complete function was soon restored to the upper extremities and some power subsequently returned to the lower extremities. There was an area of anesthesia in the 4th and 5th lumbar and 1st sacral dermatomes on the left, and bladder control was not established. For 9½ years following the procedure the neurological status remained essentially unchanged, and with the aid of crutches and braces she was able to walk and lead a relatively normal if somewhat limited life. She was subject to chronic urinary tract infection and during a severe exacerbation died of renal failure at the age of 10 years.

Pathology. The entire tumor weighed approximately 60 gm. Its uppermost pole, that lying in the region of the foramen magnum, was gray in color and microscopically was composed of necrotic, acellular material. The main body of the tumor was of a deep red color, firm consistency, and histologically contained derivatives of all three germ layers (Fig. 4). The specimen removed at the last operation was an ov-

shaped, cystic mass, most of which was covered by skin. There was no evidence of malignancy. Diagnosis: Teratoma.

Autopsy. The cord and its membranes were easily removed from the vertebral canal except in the lumbar region where the cauda equina and dura were involved in an enlarged fibrous mass. This mass was dissected free of the walls of the canal and the dura subsequently removed from about the cord (Fig. 5). Sections were cut from the cord at representative levels, and on gross and microscopic examination no tumor tissue was found (Fig. 6). At the conus medullaris a 0.8 cm. rounded, darkly discolored area presented posteriorly. On section, this area proved to be a cyst along the walls of which were several silver clips (Fig. 6D). The roots of the cauda equina

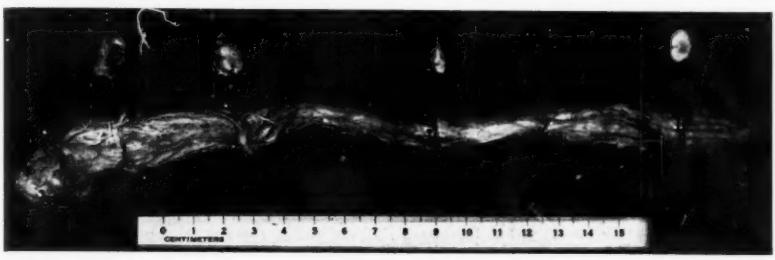

FIG. 5. Case 4. Spinal cord showing levels from which sections were removed.

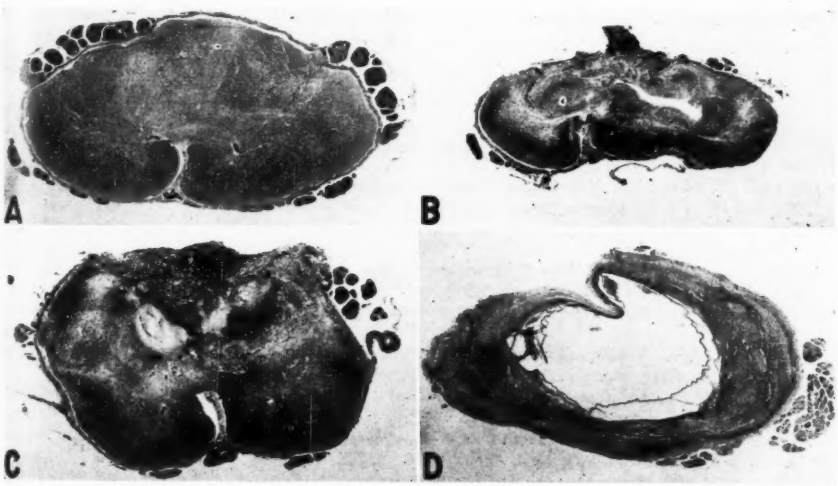

FIG. 6. Case 4. (A) Cervical region of spinal cord, showing gross architectural arrangement. At this level the tumor had been extramedullary. (B) Thoracic region, showing scarring about posterior aspect and internal derangement. In this region the tumor had been intramedullary. (C) Lumbar region. There is some scarring posteriorly; no tumor tissue remains. (D) Section through cyst in conus medullaris. In this region the intramedullary portion of the teratoma joined with the part that lay among the roots of the cauda equina. (Loyez myelin stain, ×6)

were involved in a 2×3 cm. firm, fusiform knob of fibrous tissue in which discrete neural elements could not be grossly distinguished.

The brain appeared normal and weighed 1140 gm. Other findings were cystitis with hypertrophy and trabeculation of the bladder, hydroureters, bilateral pyonephrosis and focal pneumonia.

Comment. By its very size this tumor for a while obscured the nature of the cause of the increasing paralysis. The repeated inability to obtain spinal fluid at lumbar or cisternal puncture, however, assumed increasing prominence as study of the child progressed, and the initial operation was undertaken with the provisional diagnosis of a tumor within the spinal canal.

SUMMARY

A review of approximately 650 intracranial and intraspinal tumors recorded at The New Haven Hospital over the past 20 years revealed that 1 epidermoid, 2 teratoids, and 1 teratoma had been found at operation within the vertebral canal. In this same series were encountered 4 intracranial epidermoids and 1 intracranial dermoid.

The classification of these tumors was discussed.

The 4 cases of intraspinal congenital tumors were presented and commented upon. In 1 case an epidermoid was associated with a skin dimple, congenital dermal sinus, partial duplication of the vertebral column in the region of the tumor, and diplomyelia. The 2nd patient had a teratoid of the filum terminale. A 3rd patient presented a teratoid of the lumbar region with telangiectasis and an overgrowth of hair in the skin overlying it. The 4th patient had a teratoma arising from an intramedullary site, which, becoming extramedullary, filled almost the entire vertebral canal.

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ROOT PAIN AND PARAPLEGIA DUE TO PROTRUSIONS OF THORACIC INTERVERTEBRAL DISKS*

J. GRAFTON LOVE, M.D., AND EDWARD J. KIEFER, M.D.†
Section on Neurologic Surgery, Mayo Clinic, and Mayo Foundation, Rochester, Minnesota

(Received for publication June 2, 1949)

AT THE meeting of this society in New York in 1944, one of us (J.G.L.)[2] read a paper on the differential diagnosis of intraspinal tumors and protruded intervertebral disks in the lumbar region and their surgical treatment. During the past five years, we have learned a great deal regarding the protrusion of intervertebral disks in the cervical and the thoracic regions of the spinal column. Many bizarre neurologic conditions have been found to be due to such protrusions and many patients can be benefited if proper therapeutic measures are applied before irreparable damage to the spinal cord has occurred.

The protrusions of disks in the cervical region have received a good deal of recognition, and many excellent papers have been published on the subject. Protrusions of disks in the thoracic region, on the other hand, have had little recognition. Bradford and Spurling[1] in their book reported a case of paraparesis due to protrusion of the 4th thoracic disk. Their patient made a complete recovery following operative removal of the protruded disk.

Young[3] of Australia reported 4 cases of protruded intervertebral disk in the thoracic region in which the diagnosis was proved at operation, and he emphasized that this lesion in the past has been given many different names. Lesions in the upper part of the region with reference of the pain to the thorax were called "pleurodynia," "intercostal neuralgia," "intercostal neuritis" and "fibrositis." When lesions in the lower part of the thoracic region produced radicular pain which projected into the abdomen and groin, intra-abdominal disease might be suspected.

In 1 of Young's 4 cases, the 3rd disk was degenerated, in 2 the 10th disk was degenerated and in 1 the 11th disk was herniated. In visiting with several neurosurgeons of considerable experience in all fields of neurosurgery including disk surgery, one of us (J.G.L.)[2] was surprised to learn that many of them had never seen a protruded disk in the thoracic region; and one or two surgeons felt that from what they knew about the lesion, it had best be left alone.

Because of the scant consideration that protruded disks occurring in the thoracic region have received, we felt it timely to call attention to the lesion and to report the experiences at the Mayo Clinic with it.

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* Read at the meeting of the Harvey Cushing Society, New Haven, Connecticut, June 2, 1949
† Fellow in Neurologic Surgery, Mayo Foundation, Rochester, Minnesota.

DATA ON 17 CASES

From 1922 through 1948 the diagnosis of protruded thoracic disk has been verified at operation at the Mayo Clinic in 17 cases. The relative incidence of cases of protruded disks in the thoracic region among all cases of protruded intervertebral disks (lumbar and cervical as well) was estimated for the 10-year period of 1939 through 1948 at the clinic. In this interval about 5500 operations were carried out for protruded intervertebral disks, and investigation disclosed that in 12 of our 17 cases the operation had been performed during these years. This gives a relative incidence of about 2 to 3 cases of protruded disks in the thoracic region per 1000 cases.

The incidence of protruded disks in the thoracic region was found to be

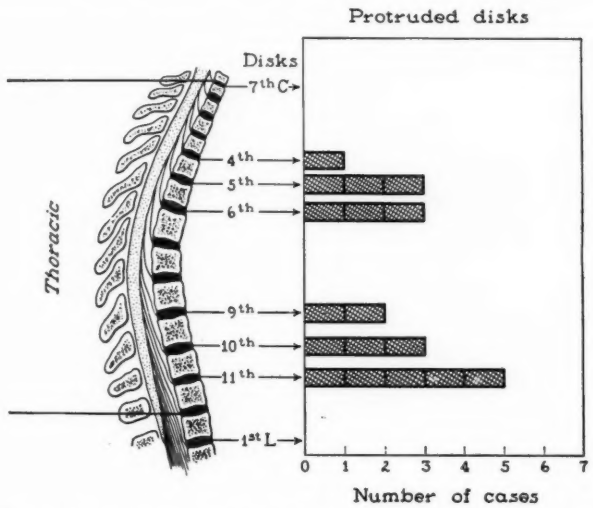

FIG. 1. Sagittal section of the thoracic portion of the spinal column showing site of the lesion in 17 cases of protruded intervertebral disk in which the diagnosis was verified at operation.

about equal in the two sexes. There were 8 males and 9 females in our series. The occupations varied from laborer to lawyer, and from farm housewife doing heavy work to stenographer. The youngest patient in the group was 26 years old. Of the other patients 7 were in the age group from 31 to 40 years; 5 from 51 to 60; 2 from 61 to 70, and 2 from 71 to 80. The oldest patient was 73 years of age.

A rather vague and unconvincing history of trauma was obtainable in 5 of the cases and in the remaining 12 no history of trauma could be elicited. The duration of symptoms varied greatly, ranging from the shortest of 3 weeks with a story of rapidly developing paraplegia to the longest of 24 years with a history of periodic lumbago.

The symptoms and neurologic examinations varied widely, depending

on not only the level of the protrusion, but the position of the protrusion in relation to the spinal cord and roots. Fig. 1 shows the relative frequency with which protruded disks were found at the various levels in the thoracic portion of the spinal column. The position of the protrusion was found to be midline in 11 cases, midline with some lateralization in 3 and lateral in 3 (Figs. 2 and 3). This accounts for the rather frequent signs referable to the pyramidal tract with all degrees of paraplegia, and the frequency of involvement of bowel and bladder as well as sensory deficits. Signs and symptoms of paraplegia were found in 10 of the cases, and of monoplegia

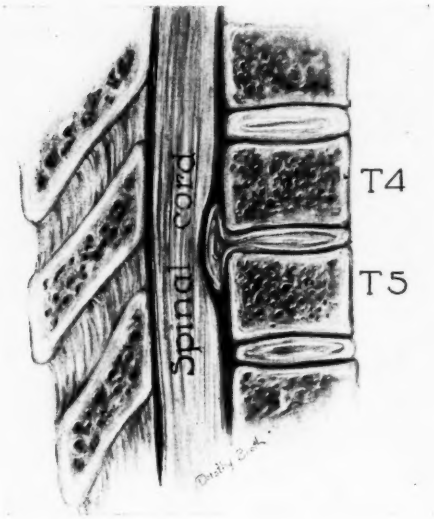

FIG. 2. Surgically exposed protrusion of the 4th thoracic disk which had produced partial spastic paralysis of the right lower extremity and subjective numbness in a woman 59 years of age. A spinal cord tumor was suspected.

FIG. 3. The protruded disk shown in Fig. 2.

in 2; neurologic examination gave negative results in 5. Dysfunction of the bowel and bladder was noted in 7 cases and sensory deficits were found in 9. Radicular or generalized pain, described as ripping, burning, constricting or pulling in nature, occurred in 12 cases. Because of the protean manifestations of protruded intervertebral disks in the thoracic region it is impossible to describe a symptom complex, such as protruded disks in the lumbar region have established.

A positive diagnosis of protruded thoracic disk was made preoperatively in only 3 cases and a tentative diagnosis in a 4th. In the 3 cases routine roentgenograms of the thoracic portion of the spinal column revealed calcification (Fig. 4) of the protruded disk and in the 4th, pathologic narrowing of the interspace was noted.

Myelograms were made on all except 2 patients; these 2 had been operated on prior to 1925. Of the 15 myelograms, 13 were made after injection of iodized oil, either lipiodol or pantopaque, and all of these except 1 revealed a defect at the level of the lesion. The myelogram that failed to reveal a defect was termed an "unsatisfactory examination." The other 2 myelograms were made after injection of air; both gave positive evidence of thoracic lesions. Results of the Queckenstedt test, which is almost routinely performed prior to myelography, were recorded in 15 cases. In 9 it gave no evidence of subarachnoid block; in 3 it showed partial block and in 3 complete block. The protein content of the spinal fluid varied from 10 to 400 mg./100 cc. with an average value of 90 mg. The cell count of the spinal fluid was normal in all but 1 case, in which it was reported as 39 lymphocytes / c. mm. A diagnosis of questionable myelitis versus a questionable tumor of the spinal cord was entertained in this case. The tentative diagnosis in 17 cases was as follows: cord tumor or cord tumor suspected in 8; indeterminate intraspinal lesion in 5; protruded calcified disk in 3; protruded disk or cord tumor in 1; myelitis or neuronitis was mentioned as alternative diagnosis in 2.

FIG. 4. Calcified intervertebral disk between the 6th and 7th thoracic vertebrae. The patient, a woman, had had thoracic pain for 9 months. The pain was not relieved by wearing a brace nor by paravertebral block. Myelogram after injection of radiopaque oil was negative for an intraspinal space-taking lesion. Total CSF protein was 50 mg./100 cc. Lateral roentgenograms revealed that the calcified mass moved at least 0.5 cm. posteriorly into the spinal canal during acute flexion of the back. The mass of cartilage was explored, found protruded and removed with relief of the symptoms.

Laminectomy was performed in all cases and the protruded portion of the disk was removed in 15. Rhizotomy was resorted to in 8 of the 15 cases to facilitate mobilization of the cord. In 2 cases decompression was performed without any attempt at removal as it was felt at the time that removal would result in too much trauma to the cord.

The follow-up study in 17 cases extends from 1 month to 11 years. Postoperative results within 1 year are shown in Table 1. In some cases motor

disability, sensory deficit and pain were not present either before or after operation.

SURGERY OF PROTRUDED DISKS IN THE THORACIC REGION

Since protrusions of thoracic intervertebral disks often compress the spinal cord and since the thoracic portion of the spinal cord occupies much more of the spinal canal in the thoracic region proportionately than the cauda equina does in the lumbar region, and since the spinal cord cannot be handled with the same relative impunity that a nerve root can, adequate exposure is essential. For involvement of a single nerve root with the protruded disk lying far laterally, hemilaminectomy gives sufficient exposure. When the lesion lies in the midline and when there is motor or sensory impairment, or both, bilateral laminectomy is advisable. Usually the lesion will extend more to one side of the spinal canal than the other. This can be determined by the neurologic findings and by palpating the lesion extradurally. Laminectomy should be extended farther laterally on the side where the protrusion is more prominent so that the lesion may be approached extradurally with the least possible rotation and elevation of the spinal cord. Usually it is advisable to divide the nerve root opposite the site of protrusion on the side on which the protrusion is to be removed. If the protruded portion of the disk is large or is centrally located, it is useful to open the dura mater and divide some teeth of the dentate ligament in order to permit the cord more freedom of movement and to lessen the traction on the cord while removing the fibrocartilaginous protrusion.

TABLE 1

Immediate Postoperative Results

	Motor Disability	Sensory Deficit	Pain
Disability increased	4	3	0
No change	4	4	2
Some improvement	5	4	3
Marked improvement	1	1	2
Complete cure	0	0	5

When the protruded portion of the disk has been removed and the spinal cord has been thoroughly decompressed, the wound should be closed in layers without drainage. If it was necessary to open the dura mater, it should be sutured water-tight with continuous stitches of non-absorbable material and then distended with physiologic saline solution. This latter procedure minimizes the danger of postoperative hemorrhage from the congested extradural vessels usually present. Fusion or bone graft usually is not required in the thoracic region.

Early motion in bed and early ambulation are advised; the patients who were not paralyzed are out of bed in 3 or 4 days after operative removal of the protruded disk. No back support is employed.

If the patient is paralyzed, physical therapy is instituted within a few days after operation and as soon as the wound is healed (about 10 days) a regular rehabilitation program is started.

RESULTS OF SURGICAL TREATMENT OF PROTRUDED THORACIC INTERVERTEBRAL DISKS

Since many of our 17 patients had severe neurologic deficit at the time of surgical treatment, the immediate postoperative results in such cases were rather discouraging. These correspond to the results obtained in the early days when patients were operated on for protrusions in the cervical region, then called "ecchondromas." Only in the late cases were compression syndromes of the cord recognized and irreparable damage to the cord often existed prior to operation. Since the syndrome of protrusion in the cervical region has become recognized, the results of operation for protruded disks in that region have improved tremendously. We think that with the increased knowledge and understanding of the syndrome of protruded intervertebral disks in the thoracic region, the results of surgical treatment in this group of cases will improve also.

FIG. 5. Calcified protruded disk can be seen at the 11th thoracic interspace. Some of the calcified material is lying posterior to the body of the 11th thoracic vertebra. Removal of the calcified protruded disk gave immediate relief of the patient's pain and disability.

If the lesion is recognized early during the stage of irritation of the nerve root or slight compression of the cord, the protruded disk can be removed without much manipulation of the cord and the patients should obtain an excellent result. An excellent example of early recognition and cure by surgical means is the case of a woman, 33 years of age, who was incapacitated by a non-radiating pain in the middle of the back of 7 months' duration. Motions in her back were markedly limited. Her chest expansion was only 2 inches (5 cm.). A roentgenogram (Fig. 5) of the spinal column revealed calcification within the 11th thoracic interspace with extruded calcified material within the spinal canal lying posterior to the body of the 11th thoracic vertebra. The total protein content of the spinal fluid removed from the lumbar region was only 10 mg./100 cc. Myelograms were not made, but laminectomy was performed. The protruded calcified disk was removed, and thus the pressure on the spinal cord was relieved. This gave immediate and lasting relief from pain and produced no neurologic deficit.

SUMMARY

Protrusions of intervertebral disks occur in the thoracic region in about the ratio of 2 or 3 to 1000 in the lumbar and cervical regions combined. A study of the records in 17 cases of protruded intervertebral disk in the thoracic region of the spinal column failed to disclose an etiologic factor in the majority of cases; a history of trauma was obtained in only 5 cases. The duration of symptoms in these 17 cases varied from rapid progression to a paraplegic state within 3 weeks of the time of appearance to 24 years of intermittent attacks of "lumbago." The symptoms and signs varied widely depending on the size of the protrusion, its location in regard to the spinal

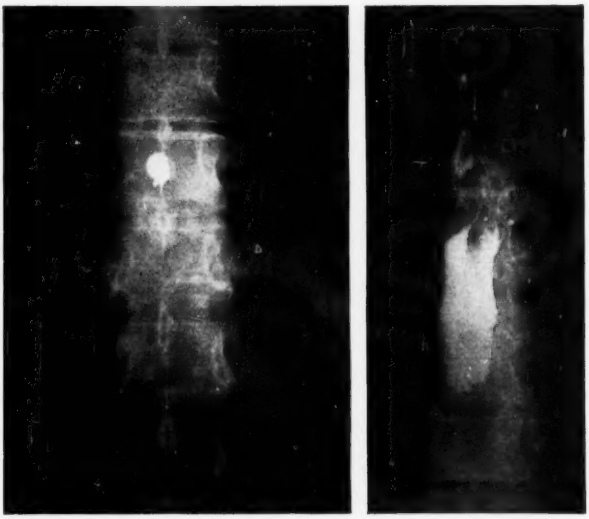

FIG. 6 (*left*). Calcified protruded disk at the 9th thoracic interspace (seen just above the head of the local anesthetic wheal needle). The patient had spastic paraplegia associated with a tight sensation around the lower portion of the chest. There was no block on lumbar puncture; total protein was 90 mg./100 cc. After removal of the protruded disk there was improvement in motor power, pain was less, but a sensory level developed at the 6th thoracic dermatome. Another lumbar puncture revealed partial subarachnoid block; total CSF protein was 160 mg./100 cc.

FIG. 7 (*right*). Same case as Fig. 6. Block of radiopaque oil at 6th thoracic vertebra. Another laminectomy was performed with complete removal of an intradural meningioma. Immediate improvement then occurred. Previously one of us (J.G.L.)[2] has called attention to the association of spinal cord tumor with protruded intervertebral disk.

column, and its situation in regard to the spinal cord. Tumor or degenerative or inflammatory disease of the spinal cord was considered in the diagnosis.

The diagnosis of a space-taking lesion in the majority of cases was established on myelographic examination of the spinal canal (Figs. 6 and 7).

Treatment was by laminectomy with removal of the protruded fibrocartilage in 15 cases and decompression of the cord without removal of the

protruded portion of the disk in 2 cases. The results of surgical treatment were brilliant only in those cases in which root pain or cord compression or both were present without gross neurologic deficit. The patients who had marked compression of the cord prior to operation usually had residual symptoms or signs indicative of irremediable damage to the cord. Thus it is evident that in order to obtain better results in these cases the condition must be suspected, recognized and treated before irreparable damage to the spinal cord has occurred.

From this study we can conclude that thoracic disks do protrude, although much less frequently than those in the lumbar and cervical regions of the spinal column. When our knowledge of this type of lesion in the thoracic region is extended and we recognize and treat it early, the results should be better than they are today.

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DRUSEN OF OPTIC NERVE SIMULATING PAPILLEDEMA

MAX CHAMLIN, M.D., AND LEO M. DAVIDOFF, M.D.*
New York City

(Received for publication July 5, 1949)

"DRUSEN" is the name applied to hyaline substances occasionally found in the papillary portion of the optic nerve. They were first described by Müller in 1858.[6] They are probably a developmental anomaly and occur as amorphous bodies of a yellowish or whitish waxy material, with or without formation of rounded lemon-yellow or golden coin-like deposits.

When these conglomerate coin-like bodies appear on the surface of the disc, they may produce a picture resembling papilledema, but, as a rule, diagnosis is not difficult. However, when they lie buried more deeply in the papilla, they may impart the appearance of papilledema, papillitis, pseudo-papilledema or pseudoneuritis, thereby producing serious diagnostic problems.

While these lesions in the disc are not common, during the past two years the authors have seen 7 cases showing well defined drusen in the optic nerve head. In addition to the 7 cases with distinctly visible drusen of the papillae 2 other cases are reported which, in the opinion of the authors, show evidence of drusen deeply hidden below the ophthalmoscopically visible surface of the papilla. This point was stressed by Reese,[7] Schlezinger *et al.*[10] and Rucker.[8]

The purpose of this paper is two-fold: (1) to review the classical picture of drusen of the optic nerve head and (2) to present criteria for the diagnosis of deeply hidden drusen of the papilla, based on our experience with these 9 cases. We had an opportunity to photograph the fundi in 8 of our cases.

RECENT LITERATURE

Since 1858, a number of articles appeared on this subject. In 1904, Cirincione[1] showed that drusen of the optic nerve do not have the same origin as drusen of the choroid. In 1923, Fuchs[2] expressed the belief that papillary drusen are derived from neuroglia cells. In 1933 Goldstein and Givner[3] observed hyalin in the neuroglia of the optic nerve. Since 1933, the literature has been rather scant on this subject. While the occurrence of optic nerve drusen without symptoms is well known, other authors describe them in association with ophthalmological diseases and still others with diseases of the nervous system. Reese[7] in 1940 showed evidence that the pathologic process is very similar to that of tuberous sclerosis and stated his belief that drusen of the optic disc are "formes frustes" of tuberous sclerosis, one of the so-called "phakomatoses."

* 815 Park Avenue, New York 21, New York.

In 1941, Samuels[9] described drusen in 20 eyes that were sectioned for microscopic study. In only 1 of the 20 cases were the drusen found on the surface of the papilla. In all the others, they were below the surface of the papilla, and this author also emphasized the point that there may be more cases of hidden drusen than are generally being recognized.

In 1944, Schlezinger, Waldman and Alpers[10] wrote an excellent article on the subject showing how drusen of the optic nerve may produce a clinical picture resembling cerebral tumor.

AUTHORS' CASES

In all of our 9 cases, whether the drusen were visible as such or not, the discs presented some of the following features. There was usually some blur-

Case No. & Eye	Visible Drusen	Blurred Margins	Yellow Pallor	White Pallor	Gray Color	Striations	Sharp Disc Margin	Fullness of Disc	Blind Spot Enlargement	Peripheral Field Defect
1 A.W.r	++	+++	0	+++	+++	+	0	+++	+	nasal
1	+++	+++	0	+++	+++	+	0	+++	+	nasal
2 G.C.r	+++	+++	+++	+	0	0	0	+++	+++	generalized
1	+++	+++	+++	0	+	0	+	+++	+++	generalized
3 S.D.r	+++	+++	+++	+	0	0	0	+++	+++	generalized
1	+++	+++	+++	+	0	0	0	+++		
4 S.E.r	0	+++	+++	0	0	0	0	+++	++	nasal
1	+++	++	+++	0	0	0	+	+	++	nasal
5 S.W.r	+	+++	+++	0	+	+	0	++	+++	nasal
1	0	+++	0	+	0	0	0	+++	+++	below
6 M.A.r	0	++	0	0	0	0	0	+++	++	generalized
1	+++	+++	0	+++	++	++	+	+	++	generalized
7 B.R.r	+++	++	+++	0	0	0	+	++	+	
1	0	++	++	0	0	0	++	+	+	temporal
8 R.B.r	0	+	0	+	+++	+++	+++	++	++	nasal
1	0	+++	0	+++	+	+	0	+++	++	nasal
9 A.L.r	0	+	0	0	+	+	+	+	++	
1	0	+++	+	0	+	+	0	+++	++	

FIG. 1.

ring of the margins and elevation of the discs. Often, there was a good deal of yellowish or whitish amorphous hyaline substance distributed within and on the surface of the disc and reaching out on to the retina, thus giving the appearance of atrophy plus blurring. With the exception of Case 2, it was noted that the yellowish discoloration was present in the older age group, and the whitish in the younger age group. Those with the whitish discoloration gave an even greater impression of atrophy than the yellow. As will be seen in Case 4, the same observer who diagnosed the condition as papilledema on one occasion called it "long-standing papilledema with atrophy" one year later.

Two of the discs showed a dirty grayish color fanning out as radial

striations into the peripapillary retina. Practically all the cases showed some fulness of the discs, even when no waxy substance was seen on the surface. The impression was that the nerve with its glial tissue was being pushed forward, as it probably is, by deeply buried drusen. Occasionally, even though most of the disc is blurred and much waxy substance is visible, a part of the disc margin may be well defined. In those cases where definitely formed drusen were seen, the discs, even ignoring the drusen, had many of the above characteristics.

Our cases were divided into 3 groups. *Group 1:* those with definitely visible drusen on both discs; *Group 2:* those with definite drusen on one disc and with ophthalmoscopic evidence of deeply hidden drusen in the other disc; *Group 3:* those with ophthalmoscopic and other clinical evidence of deeply hidden drusen in both discs.

In Group 1, the drusen were easily seen and offered no particular difficulty in diagnosis. However, even in this group, some of the observers considered them as a form of papilledema and did not recognize the drusen as such. In Group 2, one eye of each patient showed definite drusen, while the fellow in each case showed many of the characteristics of discs with drusen but not the definitely formed drusen themselves. Therefore, those eyes without the visible drusen must have had them below the papillary surface. In Group 3, 2 cases are presented. Here we found many of the characteristics of the discs in Group 1 and Group 2, but no visible drusen were seen on the surface of any of the papillae.

Group 1. Ophthalmoscopically visible drusen on both discs

Case 1. A.W., 26-year-old white male, with long-standing history of headache. X-rays of the skull and neurological examination were normal. His fundi showed drusen of both optic discs. Visual fields showed enlarged blind spots and nasal defects (Fig. 2).

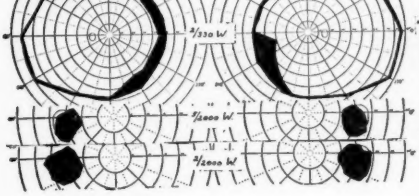

FIG 2.

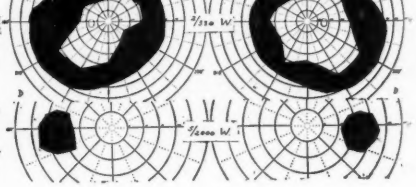

FIG 3.

Case 2. G.C., 31-year-old white female, also with a history of headache for many years. Neurological examination and x-rays of the skull revealed a completely normal

status except for drusen of the optic discs. There was generalized contraction of the visual fields and both blind spots were enlarged (Fig. 3).

Case 3. S.D., 45-year-old white male, with no symptoms referable to his eyes or head. The drusen were discovered on routine examination. The right visual field showed enlargement of blind spot and peripheral field defect (Fig. 4). Unfortunately, fundus pictures could not be obtained in this case.

Group 2. Ophthalmoscopically visible drusen in one disc

Case 4. S.E., 50-year-old white acromegalic female. Drusen were found on the left disc and evidence of subsurface drusen on the right one. The visual fields (Fig. 5) showed no evidence of chiasmal interference, but enlarged blind spots and some peripheral contraction of the lower nasal fields.

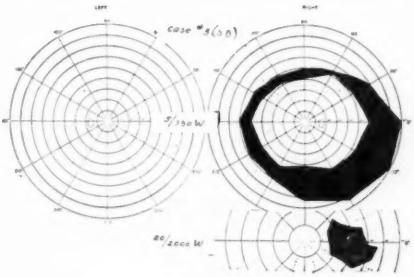

FIG. 4.

Case 5. S.W., 46-year-old white female, with complaints of headaches. Since 1939, she had been completely investigated by various observers on a number of occasions. A diagnosis of long-standing papilledema was made and used as evidence of a brain tumor to account for her headaches. She received a total of 65 roentgen treatments over scattered intervals during a 7-year period. In 1946, she was referred to us for consultation. Both discs were found to have many of the characteristics of subsurface drusen. The optic nerve heads had a yellowish waxy appearance with definitely blurred margins. In addition, the right disc showed a few coin-like excrescences on its surface. Despite the so-called "papilledema" in 1939, at the time of our examination in 1946 there was no neo-vascularization of the disc as one might expect in long-standing papilledema. In 1944 one ophthalmologist made a diagnosis of papilledema and, in 1945, the same ophthalmologist recorded the appearance of these discs as "blurring with atrophy." This ophthalmologist was kind enough to read his findings from his records to the authors, and the description of the disc margins and color in 1944 and 1945 were practically an exact description of what we saw in 1946, and again in 1948. In other words, this was a picture that resembled papilledema with atrophy but did not change for at least 5 years and probably longer. Our visual field studies (Fig. 6) revealed enlarged blind spots and some peripheral contraction which also did not change over a period of 2 years while under our observation.

Case 6. M.A., 19-year-old white female, admitted to the hospital October 1946. The patient complained of headaches for 6 months. A complete neurological examination, including pneumoencephalography and electroencephalography, was not remarkable. The left fundus revealed distinct drusen on the optic disc, with blurring of the margins by a whitish waxy material, some of which extended into the retina around the macula. The right fundus showed blurred margins and fulness of the disc. The visual field studies (Fig. 7) showed enlargement of both blind spots and some irregular peripheral contraction.

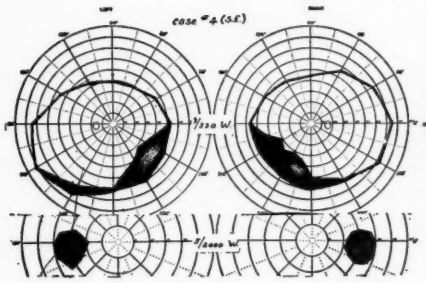

FIG. 5.

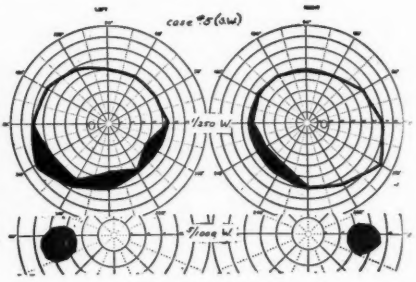

FIG. 6.

This patient was seen again a year after her discharge from the hospital with no complaints and no new findings.

Case 7. B.R., 45-year-old white female. In 1944, on routine ocular examination for refraction purposes, drusen were discovered in the right optic disc. Both discs showed blurring of the margins by a yellowish waxy material. The visual field studies (Fig. 8) revealed enlarged blind spots and some peripheral contraction. Up to the present, 1949, none of these findings has changed.

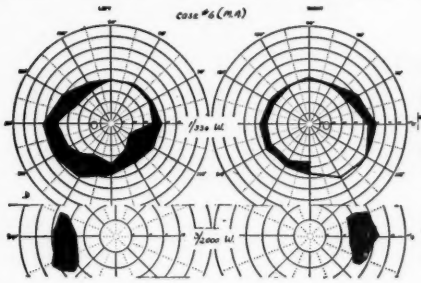

FIG. 7.

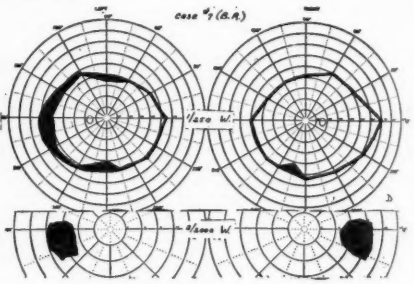

FIG. 8.

Group 3. Subsurface drusen in both discs

These patients, although showing no visible drusen, were believed to have deeply buried drusen within the papilla. The diagnosis was made by the ophthalmoscopic findings, the history, the visual field changes and the exclusion of increased intracranial pressure by neurological and laboratory investigations.

Case 8. R.B., 14-year-old white female, admitted to the hospital on April 23, 1947 for what was thought to be papilledema. *Right eye:* the disc showed some grayish peripapillary striations all around, except at the temporal border where the disc margin was well defined. The upper and lower nasal portions of the disc were elevated and appeared edematous at the margins. Despite the apparent edema involving three-fourths of the circumference of the disc, the temporal margin remained sharp and the physiological cup was still present. The vessels were not engorged and there were no haemorrhages or exudates. *Left eye:* the entire disc appeared blurred, with a

hazy whitish material extending into the circumpapillary retina. Again, a very small sharp temporal border was visible, and the vessels were not engorged. Visual field studies (Fig. 9) showed very definite enlargement of both blind spots, together with some irregular peripheral contraction, a little more marked below and nasally. Because of the lack of central and paracentral defects, the good corrected visual acuity, and the lack of perivascular exudates or changes in calibre of retinal vessels, optic neuritis was discounted. In view of the persistence of some clear disc margins with so much apparent edema and with a history of 10 days' duration and the lack of haemorrhages or engorgement of the retinal veins, papilledema due to increased intracranial pressure was considered unlikely. Since the blind spots were enlarged, the diagnosis of "pseudopapilledema" was discounted.[4]

For these reasons, a diagnosis of drusen of the optic discs was made, and it was believed that they were hidden below the surface of the papillae. Complete neurological investigation including pneumoencephalography and electroencephalography showed no abnormal findings. Reexamination in 1947 and again in 1949 revealed no changes in the ophthalmoscopic or visual status.

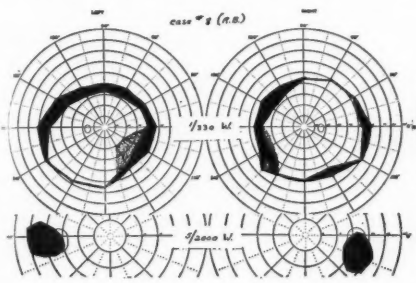

FIG. 9.

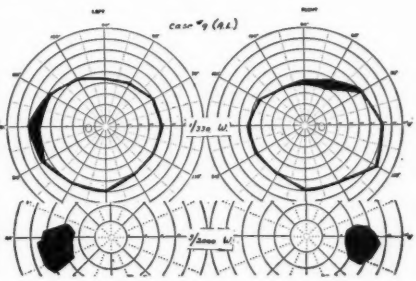

FIG. 10.

Case 9. A.L., 45-year-old white female. In March 1948, the patient complained of blurring in the left eye. She was examined at a hospital on April 1, 1948, and was told she had "papilledema." She left the hospital and went to a neurologist who thought it more likely that she had optic neuritis.

On May 21, 1948, she was admitted to our service for investigation, and it was found that the "blur" in the left eye was correctible with a +1.00 sphere. Both blind spots were definitely enlarged (Fig. 10). The peripheral field of the left eye showed a mild irregular contraction. Both discs appeared full with a yellowish undertone. The left disc showed definite yellowish blurring of the margins, while, in the right disc, the margins were still easily visible. In both fundi, there was a dirty gray color with striations coming off radially from around the disc margins. Neurological examination, electroencephalography and roentgenograms of the skull were all normal.

DISCUSSION

In all the cases in this series there was a definite enlargement of the blind spots, and such enlargement has been described by some of the observers mentioned above.[7,8,9,10]

This is readily understandable when one examines the sketches and

microphotographs published by Samuels,[9] who made a histopathological study of 20 globes with drusen of the optic disc. In this study, the drusen were seen to encroach upon the peripapillary percipient elements pushing aside the peripapillary rods and cones. This would account for an enlargement of the blind spots in the same way as is produced by papilledema.

The peripheral field changes described have been diverse in character. On purely theoretical grounds, many types of defects can be found, depending on the number and distribution of drusen and the amount of interference they cause in the nerve fibre bundles in their proximity. Rucker[8] reported a few diverse types he encountered with papillary drusen and concluded that there may be arcuate defects, either small or breaking through to the periphery, or peripheral contraction, mostly nasal and below.

In our series of cases, while most of them showed rather irregular peripheral contractions, the most numerous were on the nasal side, and some of the deepest indentations from the periphery were below and nasal (Cases 1, 4 and 8). However, we do not believe any practical diagnostic importance can be ascribed to these particular field defects, since they depend on the fortuitous distribution that the hyaline bodies happen to have.

While 5 of the 9 patients in this series complained of headache, and various authors have attempted to show that headache may be an accompaniment of this condition, we must remember that people with headache from any cause are more apt to come to the attention of neuro-ophthalmological observers. We do not know how many people with papillary drusen and without headache never come to the attention of the neuro-ophthalmologist.

From the viewpoint of the clinical neurologist and ophthalmologist, the most important significance of these drusen lies in their resemblance to papilledema. Four of our cases had been seen by ophthalmologists and neurologists who labelled them as papilledema.

Less frequently, optic neuritis and pseudopapilledema may be confused with this condition. The visual field changes, however, more closely resemble papilledema than papillitis, because of the preponderance of peripheral rather than central defects.

So-called "pseudopapilledema" should not show any enlargement of the blind spots.[3] However, until definite histological studies of discs with pseudopapilledema are made, we may even suspect the possibility that such discs have deeply buried drusen not reaching out enough to encroach on peripapillary percipient elements and therefore presenting a normal blind spot. The possibility of so-called "pseudopapilledema" really being cases of deeply buried drusen has also been mentioned by several of the authors quoted by us.[9,10]

The absence of a sudden onset of central and caeco-central scotomas will usually rule out papillitis, although nerve fibre bundle defects can occur in both.

A very important step in evaluation is the static nature of the process.

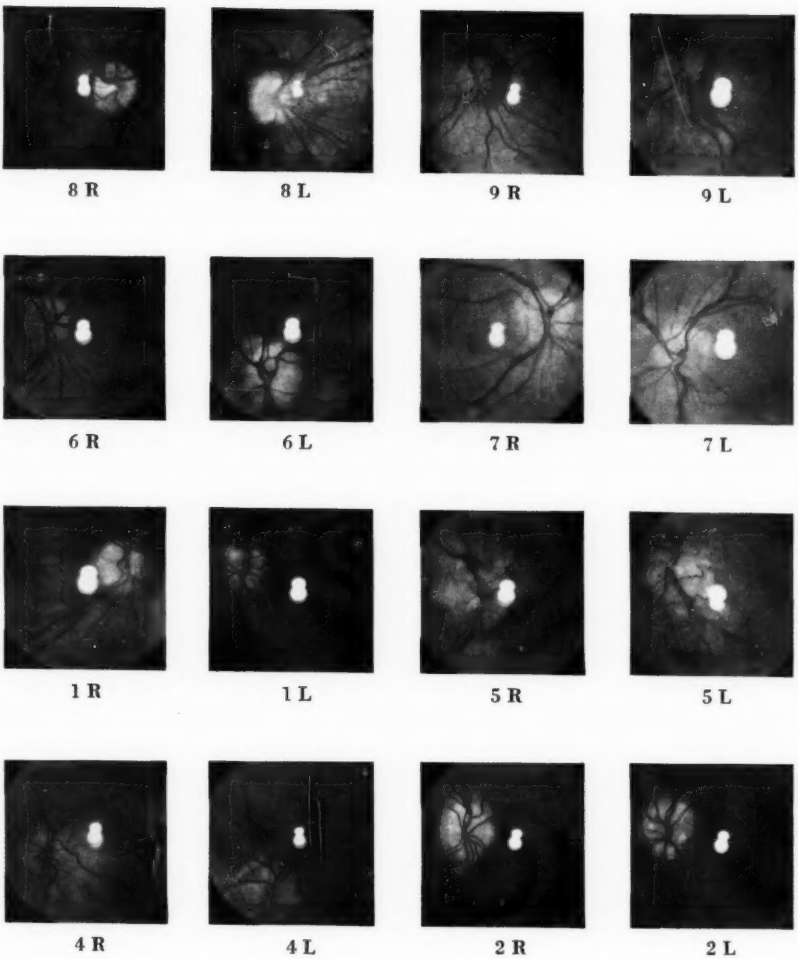

FIG. 11. Points of resemblance of cases in Group 3 (no definite drusen visible on surface) to the other discs, with drusen visible or drusen in fellow eye.

Case 8. Left eye: looks very much like left eye in Case 6 except for visible drusen in the latter. Right eye: nasal margin is whitish and blurred, like most of Case 6's left eye and, even more, right eye.

Case 9. Left eye and right eye: this is an older patient, and the whole fundus is consequently a little more yellowish. The color is very much like that of right eye in Case 4, as well as left eye in Case 4, except for the actual drusen in the latter. The two discs also resemble that of left eye in Case 7, as well as right eye of Case 6, although the latter is a younger patient and, consequently, has a less yellowish or more whitish fundus and disc.

While increase in size and number have been described by Walker,[11] Lauber,[5] and Reese,[7] this growth is very slow and may take place over a number of years.

True papilledema is not likely to last for long periods of time without showing haemorrhages, exudates, venous engorgement, etc. Even if it is low grade and the venous system can accommodate itself to the increased intracranial pressure without becoming engorged, one would expect secondary vascularization on the disc and much more pronounced field changes from the inevitable optic atrophy that must accompany long-standing papilledema.

There may be, however, mixed pictures of recurrent papilledema with underlying atrophy that can simulate drusen of the nerve head. In such cases the patients will have had papilledema for a long time and will, in all likelihood, show much more clinical evidence of increased intracranial pressure.

For these reasons, it is important to obtain previous records of funduscopic examinations. In cases such as these, the authors make every attempt to obtain a history of a previous ocular examination by an ophthalmologist. A record by a colleague indicating blurring of discs or a yellowish appearance 3 or 4 years before could be of great help in reaching a decision as to whether one is dealing with hidden drusen or true papilledema. It is evident how important it is for ophthalmologists to make a few notes as to the appearance of all discs, even normal ones, on routine examination, with particular reference to the disc margins, the cup, the color and calibre of vessels.

SUMMARY AND CONCLUSIONS

Drusen of the optic discs occur as a developmental anomaly and may cause a picture resembling papilledema or papillitis. When lying on the surface as discrete, coin-like bodies, they are easily recognizable as such ophthalmoscopically. When buried below the surface of the papilla, they may so closely resemble papilledema or papillitis as to cause a serious diagnostic problem.

If one sees discs that are swollen, blurred at the margins and having a yellowish or whitish waxy material and perhaps a grayish discoloration off the disc; furthermore, if the veins are not engorged and no haemorrhages or discrete exudates are found in the peripapillary retina, and, if the blind spot is enlarged but the rest of the clinical picture does not suggest increased intracranial pressure, the diagnosis of deeply hidden drusen of the optic nerve must be considered.

If in doubt, and the presenting symptoms are not urgent, it is worthwhile following the case with funduscopic study and blind-spot plotting over a period of weeks or even months. Papilledema should certainly show some remarkable fluctuations both in ophthalmoscopic appearance and blind-spot size, while drusen will show little or no changes.

If symptoms appear to be acute, one may have to do pneumoencephalo-

grams. If the gas studies are normal and the neurosurgeon convinced of the absence of increased intracranial pressure, one may assume the presence of drusen of the optic nerve.

The authors do not imply that all cases with deeply hidden drusen can be diagnosed clinically with ease. Surely, many cases will need gas studies, angiography, and other forms of neurological investigation to satisfy the neurosurgeon.

Again, once such diagnosis is made, the patient should be followed at reasonable intervals for a year or so, during which time the fundi and blind spots should be studied comparatively.

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EXPERIENCE WITH TOPECTOMY FOR THE RELIEF OF INTRACTABLE PAIN*

J. LE BEAU, M.D.†
Paris, France

(Received for publication July 6, 1949)

INITIATED by Pool[4] in 1946, topectomy in his hands has been a less mutilating and as efficient an operation as bilateral leucotomy in the treatment of many lasting mental disorders. Bilateral topectomy of the region of areas 9, 10, and 46 appears to produce an especially marked effect on the anxiety of patients. This fact, and the reports of some good results obtained by Freeman and Watts[3] and others using bilateral leucotomy in cases of intractable pain, led me to try bilateral topectomy in such conditions. It was hoped that intractable pain could thus be relieved without mental deterioration. The first results were reported in 1948.[6] Those early cases, in which operation was performed in 1947, and more recent ones, are presented here, making a group of 25 patients with intractable pain submitted to bilateral topectomy.

OPERATIVE TECHNIQUE

The details of the operative technique have been described before[5] and will therefore be omitted here. However, there are certain points to which attention may be called. The first is the actual extent of excised cortex. As a rule, we remove a cortical zone starting 5–6 cm. in front of the coronal suture, and 3–4 cm. long, ending 1–2 cm. from the medial part of the orbital roof. Laterally it extends 3 cm. from the medial line (a little more on the right side than on the left). Medially it is between 1–1½ cm. down along the falx. The average weight of our specimens was 10–12 gm. on each side; that is, about half as much as Pool's removals, which extend definitely deeper (2 cm.) on the medial aspect of the hemisphere. In that respect, it is important to state again that, as a rule, we are not dealing here with mental cases, where 20–25 gm. on each side seem to be the measure of cortical removal necessary. Rightly or wrongly, in our patients with pain a more limited removal and possibly a more specific effect were looked for.

As prefrontal lobe surgery is still partly in the research stage, it is necessary to check the extent of excised cortex by taking postoperative roentgenograms; if care is taken to place Cushing clips on the limits of the cortical resection (without putting any on the dura) a fairly precise evaluation is obtained. This can be very useful when, as happened in 2 cases, recurrence of pain apparently is related to too limited a resection.

In Figs. 1 and 2, we would say that there were excised the rostral half

* Since this paper was received, the follow-up notes have been brought up to date of September 1949.

† Neuro-chirurgien des hôpitaux de Paris. Address: 107, Rue de l'Université, Paris, 7e, France.

of area 9, the dorsal two-thirds of area 10, and the medial third of area 46. This calls for an explanation of what is meant here by these area numbers: they are used merely as topographical landmarks and for brevity of language. For instance, instead of saying that one excises a region of prefrontal cortex within such and such distance in cm. from the orbital roof, the Sylvian fissure, the callosal fissure, etc., we find it more convenient to say that one excises "area 10." This is a short term for area 10 as depicted in Brodmann's classical map, though we do not believe that we excise with precision delicately defined architectonic areas. We are to be blamed in using area num-

FIGS. 1 and 2. Case 10. Side and front views of bilateral topectomy.

bers in such a loose sense, but we find it a very simple way of describing the operation.

POSTOPERATIVE COMPLICATIONS

Thirty operations were performed on 25 patients, with 2 postoperative deaths: 1 from acute lung edema (carcinoma of lung), and 1 from vascular collapse (thalamic syndrome and high blood pressure in patient aged 65). Indeed, many patients with intractable pain are poor operative risks, and especially in those over 60 with arterial disorders, unilateral lobotomy, as proposed by Scarff,[9] should be tried first, in our opinion.

Neurological complications were rare. In 2 patients, one epileptic attack occurred the night following the operation, without recurrence. For the time being, none of them has epilepsy; none of them takes luminal or dilantin. In 1 case (thalamic syndrome, Case 15), motor aphasia lasted for 2 days. The longest follow-up period has been 23 months.

Sphincteric disturbances (incontinence) were not always met; when they

did occur, they persisted less than 1 week. Sometimes they lasted between 4 and 6 days, as in Case 6 (resection of 10 and 11), Case 14 (thalamic syndrome), and Case 19 (tabes dorsalis). Eventually bladder function always became normal. In that respect, the importance of good nursing should be stressed; patients as a rule should get up no later than the 10th postoperative day and start walking 1 or 2 days later.

Postoperative mental disorders were met in nearly all cases; in two-thirds of them, it was a mild indifference and apathy, which cleared completely after 6 or 8 days. In one-third, some agitation persisted for 2 or 3 days but was not difficult to control without morphine. A typical "frontal syndrome" lasting 2 and 4 weeks was observed in Cases 2 and 19. Finally, in Case 4, which turned out eventually to be a failure, there was mental confusion for 2 months (the patient was an old woman with a history of mental disorders in the family).

The problem of the definitive action of topectomy on the normal or abnormal pre-operative mental condition will be discussed later.

ACTION OF TOPECTOMY ON PAIN

1. *General Results.* The results of the operation are summarized in Table 1. The effect on pain is expressed by the following scale: 0 means no change whatever occurred in the patient's experience of pain; 1 means suppression of sedatives such as morphine, although the patient still complains; 2 means that the patient no longer behaves as if he still had pain, but nevertheless states that he has pain when asked about it; finally, 3 means the patient himself says he has no more pain, or only occasionally has pain.

From a practical point of view, it must be borne in mind that not only grade 3, but grade 2 as well, are equivalent to a very good result, next to a complete cure. In grade 2, the patients exhibited three improvements in objective behavior, which are more important to us than psychological tests. These patients sleep again without sedatives; they eat again and gain much weight; they work again. Furthermore, relatives are satisfied that the patients are no more worried by pain. Finally, even when the patient states after questioning that he still experiences pain, never does his facial expression betray it, and his behavior remains calm and composed.

Thus it is believed that practically complete cure was achieved in patients classified 2 and 3. We have 19 such cases out of 25. But, as can be seen in Table 1, for most of them the follow-up is not long enough, and all that can be said is that bilateral topectomy has rid them of their pain to date. In 11 cases (Nos. 1, 2, 3, 5, 6, 8, 9, 10, 11, 18, 22), good results have lasted more than 8 months, and, in 2 instances, almost 2 years. On the other hand, it should be noted that failures always become evident within less than 3 months. There were several such patients, and a few were submitted to another intervention, as will be discussed in the following paragraphs.

2. *Failures and Possible Causes.* One reason for failure might be when one has tried to deal with an evolutional disease by topectomy. This appeared

TABLE 1

Topectomy and intractable pain (25 cases)

Case No. and Diagnosis	Result on Pain	Postoperative Follow-up (in months)
NEURALGIAS—7		
1. Atypical Facial	2	23
2. Atypical Facial	2	22
3. Atypical Facial	2	16
4. Atypical Facial bilateral	0	(3)
5. Occipital	3	8
6. Hemicranial	3	8
7. Perineal	1	(1 week)
NERVE INJURIES—6		
8. Cervico-occipital (x)	3	16
9. Occipital	3	19
10. Causalgia	3	8
11. Causalgia (x)	2	8
12. Phantom Limb	3	6
13. Painful Stump	2	4
VASCULAR AND INFECTIOUS NERVOUS DISEASES—6		
14. Thalamic Syndrome	?	Postop. death
15. Thalamic Syndrome	1	2
16. Thalamic Syndrome (x)	3	1
17. Encephalitis	0	(3)
18. Tabes Dorsalis	3	8
19. Herpes Zoster	3	3 weeks
CARCINOMAS —6		
20. Rectum	3	2
21. Stomach (L)	3	8
22. Lung (L)	?	Postop. death
23. Uterus (L)	0	(1)
24. Spine (x)	3	1
25. Tonsil (x)	0	(1)

(L) = Unilateral left topectomy.
(x) = Two operations performed for recurrence of pain.
In the follow-up column, numbers in parentheses express when failure became apparent.

to be the explanation in Case 17, where violent and nearly permanent occipito-cervical and brachial pains were checked for only 2 or 3 months after operation. After that, signs of evolutional encephalitis with alterations of the cerebrospinal fluid (without germ) were observed with periods of amelioration; one phase of mental confusion appeared 3 months after operation and then cleared up. Later, with a better psychic state, the patient complained of violent pains as before, but they were not so lasting. *This evolutional factor may explain, too, why long-term results are doubtful in cases of rapidly growing carcinoma.* On the other hand, intermittent attacks of violent pain, as in Case 18 (tabes dorsalis) and Case 10 (causalgia with both permanent pain and paroxysm), were checked after bilateral topectomy. In

any event, the problem of how a topectomized patient would react to a new and permanent cause of violent pain is not yet solved.

It is possible to explain two other failures we observed, by the contention that in these cases neuralgia was associated with a definitely abnormal preoperative mental state. In Case 7, the patient was an old man with marked hypochondria for more than 20 years; the only postoperative result was the suppression of his drug addiction. In Case 4, the patient was an old woman with hereditary and familial evidence of indefinite mental disturbances; she suffered from a bilateral permanent facial neuralgia, and topectomy brought about improvement for less than 3 months.

Finally, other failures may be due to an insufficient cortical excision. In Case 23, (pelvic carcinoma, sciatic pain, morphine addiction), *unilateral left topectomy* proved futile, since within 1 month both pain and morphine addiction reappeared. The patient then refused a right topectomy. In Case 11 (causalgia) and Case 24 (carcinoma of the spine), unilateral left topectomy was tried first, with recurrence of pain within about a month. Right topectomy was then performed with good results (1 month in Case 24, after which death occurred as the disease progressed; 4 months to date in Case 11). But one very good result was achieved in a case of left topectomy (Case 21); coeliac pains disappeared for 8 months, until the time of the patient's death. On the whole, unilateral topectomy seems not so good as unilateral lobotomy, if Scarff's results are confirmed. In Case 16 of our series, a left lobotomy did not rid the patient of his complaints, and a right topectomy was then successful. However, the postoperative follow-up on this patient is too short to be certain of results.

Insufficient cortical excision is the reason for failure in some cases of bilateral topectomy. We had 2 such cases (8 and 25).

In *Case 8*, which is the more interesting, there developed a depressive state, which was treated by electric shock; the patient then complained of unbearable permanent occipito-cervical pain. Believing in the existence of a vertebral fracture, a surgeon operated on this patient, and a posterior cervical radicotomy was performed. The situation grew worse and worse, even after performance of a bilateral prefrontal leucotomy, which was probably incomplete. When we first saw the patient, she presented a mixture of anxiety, persecution, and constant complaints of occipito-cervical pain. On Nov. 10, 1947, bilateral topectomy brought about a definite improvement, but 2 months later, recurrence of pain and the anxiety state was obvious. On examining the resected cortical areas, it was found that their weight was only 6 gm. on the right side and 5 gm. on the left, and it was judged that removal had not extended far enough from the medial line.

On May 1, 1948, at re-intervention, area 46 on the left side was undercut, and on the right side its medial third and the dorsal half of area 10 were excised. From that day on, marked and progressive improvement was observed, and when last seen a year later, the patient was judged mentally normal. She no longer complained of pain but stated that her neck was still stiff (which indeed it was). She had gained much weight and for the past 6 months had again been able to take up her work as dressmaker with good success.

In *Case 25*, the patient had a tonsillar carcinoma with violent permanent pain and unbearable attacks of pain when he tried to eat. On Nov. 11, 1948, bilateral topectomy was performed, with great improvement which lasted 3 months. Then the situation progressively returned to its former state. Here again, cortical fragments were found to be very small; about 5 gm. had been removed on each side, and they looked too narrow (under 15 mm.). Comparing this with Case 11, a re-intervention was then decided on, and on April 21, 1949 more cortex was excised on each side, extending over area 46, 30 mm. on the left side and 35 mm. on the right side from the medial line. The patient was better for 1 month, then he complained again while his carcinoma grew very rapidly; death occurred 3 months later.

It would therefore seem that 5 or 6 gm. on each side is definitely not enough to achieve lasting results, although as in our Case 9, that amount of cortical resection gave a very good result lasting 15 months to date. When, on the other hand, it is noticed that in one of our definite failures (Case 17) the amount of resection was 15 gm. on each side, the only reasonable conclusion is that it is too soon to tell whether the action of topectomy on pain is a quantitative effect. It can only be stated that the best results were obtained on the average with a weight of 10 to 12 gm. on each side, when care was taken to excise as far as 30 mm. laterally (medial third of area 46). The problem of localization will be referred to again in this article.

RESULTS OF TOPECTOMY IN RELATION TO DIFFERENT CLASSES OF PAIN

Three classes of pre-operative pain are recognised: (A) Pain with no known organic lesion, and as a rule with an abnormal mental state, (B) pain with a definite organic nervous lesion, and a normal mental state, and (C) mixed cases.

If we refer again to Table 1, it will be seen that the Neuralgia group belongs to Class A, whereas the other 3 groups belong to Classes B and C. In Class B we place Cases 10, 18, 19, 20, 21, 24, 25; in Class C, Cases 8, 9, 11, 12, 13, 15, 16, 17, 23.

A. *Pain Not Related to Any Known Organic Lesion.* This class comprises the 7 patients with neuralgia, and all of them were in an abnormal mental state before the operation. Two had definite mental disorders: Case 7 (hypochondria), with a result of nearly complete failure, and Case 5 (obsessive compulsive), with a good result for pain and no mental improvement. The other 5 patients were mostly in anxiety states developing at the same time as permanent pain. Case 4 is a failure, and another intervention is contemplated. (Unfortunately, the first cortical fragments were lost and not weighed, but the operative description leads one to believe they were too small.) Cases 1, 2, 3, and 6 were very good results and lasting ones. Case 2 is the most demonstrative one, since the patient, a skilled worker, is back at his former job, which had been interrupted for 1 year before topectomy. The others are housewives and lead a normal life. These 4 patients are not only rid of their pain, but they are rid of their anxiety as well, and whatever the final explanation may be, it is a fact that *there was a close parallel between*

the establishment of anxiety and unbearable pain, and between their disappearance following bilateral topectomy. This may have a physiological significance.

Practically, the so-called "psychalgias" seem eminently suitable for topectomy. Of course, peripheral nerve sections are futile, though it is sometimes difficult to tell in advance. Indeed, our patients, 1, 2, and 3 were submitted to trigeminal root section and then exhibited the condition known as anesthesia dolorosa. Bilateral frontal lobotomy is likely to work, but topectomy has the great advantage of not leaving any significant mental deterioration. We do not know if unilateral lobotomy gives lasting good results in such cases.

Incidentally, the use of the word "psychalgia" or "psychic neuralgia" should be discussed. It is not a bad expression if it means that the responsibility for unbearable pain lies in the central nervous system, *not* in the spinothalamo-parietal circuit. But if it means that this pain is something akin to hysteria or to a disorder of the patient's imagination, this does not make physiological sense to us. It is suggested that the word *souffrance* should be used, as it conveys the impression of a mixture of organic pain and anxiety. *Souffrance*, it is hoped, may be submitted to some sort of physiological analysis, as will be seen presently.

B. *Pain Related to a Definite Organic Lesion, with No Significant Preoperative Mental Change.* This group is made up of 4 patients with carcinomas (Cases 20, 21, 24, 25) without morphine addiction; 1 with causalgia (the patient was on 1–2 gr. morphine daily but did not show at all the toxicomanic mentality); 1 tabetic with 3 or 4 weekly attacks of very severe pains in the legs; and finally, 1 very distressing case of ophthalmic neuralgia following herpes zoster.

All results but 1 in this group are listed as 3, meaning that they are the best possible. But it must be stated at once that in 4 cases the follow-up did not last more than 4 months. Three patients with carcinoma were completely relieved up to the time of their death, and this was 8 months in 1 case. The 4th case was a failure.

Non-malignant diseases are of course more interesting in measuring the value of topectomy against unbearable organic pain. The case of causalgia (No. 10) lent itself to some physiological study of unbearable pain, or *souffrance*, as described in the following report.

Case 10. A male, aged 47 years, in a car accident on June 25, 1948, injured his left forearm with destruction of skin, muscles, brachial artery and median nerve. One month later, a typical causalgia appeared with both permanent pain and unbearable paroxysms, either spontaneously or after the slightest touch. Morphine was necessary. The pain soon invaded the precordial area with anginoid feeling; the left hand exhibited classical vasomotor disturbances, and no movement whatever of the left wrist was possible. On Sept. 29, 1948, excision of the stellate ganglion and the first 2 thoracic was performed. There was improvement for 3 weeks, and then the pain recurred. Also, a hypersensitivity of the ulnar nerve below the left elbow developed progressively and grew worse after an operation aimed at freeing the nerve from a bone fragment. When the patient was seen in January, 1949, his con-

dition was pitiful, but it must be stressed that, although morphine was necessary, he exhibited no anxiety other than what was related to his pain. He was a perfectly normal man before the injury and held a responsible position in a telegraphic organization.

This patient seemed an ideal case to test any objective change in unbearable pain after the operation, because sensitivity and reaction to pain could be explored over the left arm by means of localized pressure (measured in grams). Fig. 3, showing the pre-operative state and the postoperative changes, speaks for itself. Two thresholds were determined: first, the patient's recognition of unpleasant pain, which

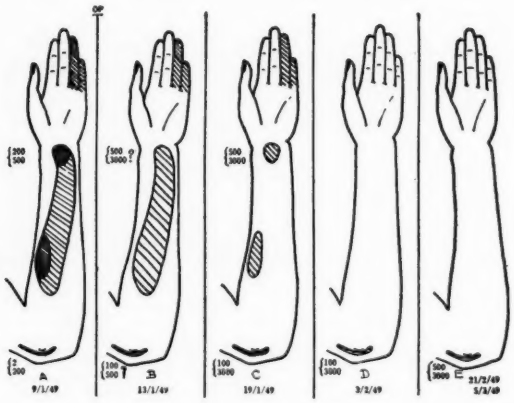

FIG. 3. Case 10. Thresholds for unpleasant and unbearable pain are expressed in grams. Operation: Bilateral topectomy.

is not the threshold of identification of pain as such but a larger one; second, the state of unbearable pain when the patient can no longer submit his forearm to investigation. The second threshold is of course even higher than the first. Admittedly, an analysis of this kind cannot be a very precise one, but the postoperative changes are so striking (compare A and E) that there is little doubt that topectomy brought about an objective change in what we call *souffrance*.

Other changes were in the surface of the reactive area (where unbearable and unpleasant pain could be elicited), which shrank down to next to nothing. Morphine was suppressed at once and ever since. Precordial pain and anginoid feeling disappeared. Anxiety progressively subsided, and the patient realized his pain was less and less marked. Finally, due to disappearance of pain, voluntary movements became possible in the left wrist, and vasomotor disturbances were improved.

Mentally, after about a week of relative indifference, the patient became completely normal again, interesting himself in his social and affective life. He flew back to Morocco 3 months later to resume his work, and up to August, 1949, the cure can still be considered as complete.

Now the follow-up of such a case is too short to be sure of a definitive cure. But this does not alter the fact that *souffrance* was objectively changed and could be roughly measured. The already noted parallel between anxiety and souffrance leads to bring them together, and they may very well be two

sides of the same cerebral function. If this is true, the above case of causalgia presents a physiological means of measuring a mental state.

From a different and practical point of view, in unbearable pain consecutive to organic nervous lesions, topectomy seems a promising operation. For the same reasons as above (Class A), we are not in favour of bilateral lobotomy, though mental changes are of lesser social importance when dealing with a rapidly growing carcinoma. Unilateral lobotomy should be considered, as Scarff reported very good results in such conditions. But unbearable pain related to an organic nervous lesion lying below the 5th cervical segment should be submitted to heterolateral cordotomy first, if the patient is not on morphine and if the pain is strictly unilateral. Walker's mesencephalic tractotomy deals with heterolateral pain in the cervical and cranial segments; in such conditions, which operation should be chosen first is still open to discussion, and bilateral topectomy is technically easier to perform.

C. *Pain Related to Organic Lesion, with Mental Abnormality.* This class includes in our series 5 nerve injuries (Cases 8, 9, 11, 12, 13); 3 brain diseases (15, 16, 17); and 1 carcinoma with drug addiction (23). In all these 9 cases, the pre-operative mental state was abnormal, and this could be related in 6 cases to an aftereffect of pain, either too long-standing, or too acute, or both. Five patients became drug addicts following the actual experience of pain. One patient had causalgia for more than 25 years; drug addiction did not develop, but a schizoid mentality became more and more marked. In the 3 remaining cases (8, 9, 17), it is likely that the preoperative mental disturbance was not directly related to pain, at least at the beginning.

Two cases were complete failures: a unilateral left topectomy (23), and an evolutionary encephalitis (17). But 6 among the 7 remaining patients did very well, and in 2 at least the follow-up seems satisfactory (16 and 19 months), the patients having resumed their work of charwoman and dressmaker. Case 11, with causalgia of the left median nerve, allowed us to perform the same experimental control as in Case 10, described above in Class B. Fig. 4 shows the pre-operative state, the changes following left topectomy, and the changes following bilateralization of the topectomy, with good results which appear stabilized by now. In the same time, the patient in whom a schizoid personality developed in the course of his 25 years of causalgia showed an important mental improvement; he gave up his isolated life, lives and talks with people he avoided before, and is ready to start as active work as possible. As in Case 10, a striking parallel is observed between the improvement in pain and the gradual disappearance of feelings of anxiety. Objectively, the result with regard to pain is not so good as in Case 10, in which duration of causalgia was much shorter. In any event, here again we feel topectomy enabled us to measure roughly a special quality of pain, called *souffrance*, and closely related to, if not the same as, a special quality of mind called *anxiety*. The same tests were performed on Case 15 (thalamic syndrome), in whom postoperatively were found much higher thresholds for bearable pressure.

Class C, together with Case 7 in Class A and Cases 10 and 20 in Class B, provide a good opportunity for studying the *action of topectomy on drug addiction*. Nine patients regularly took morphine, or Sedol, or Demerol before the operation, but only 6 exhibited the general features of drug addiction. If we exclude Case 23, where unilateral topectomy led to a failure, it will be seen that in all cases during the hour following operation, morphine or Sedol or Demerol were suppressed abruptly. Only luminal, and sometimes chloral, was given for a few days. None of the 8 patients ever asked for his drug again, and none of them showed any significant withdrawal symptoms. This means that some agitation could be observed sometimes for 24 or 48 hours, but it

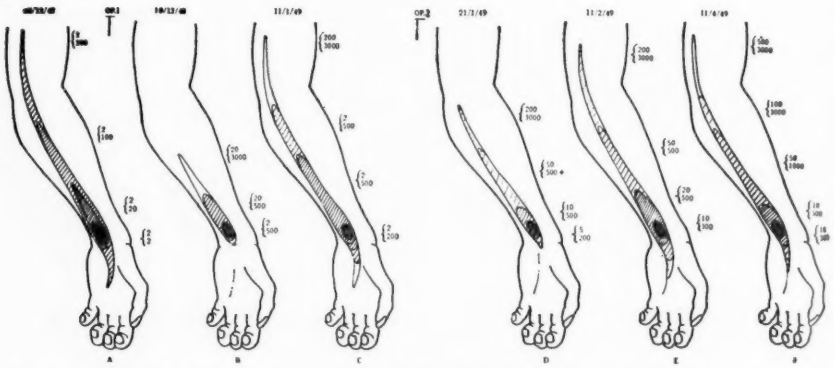

FIG. 4. Case 11. Thresholds in grams as in Fig. 3. Op. 1 = Left topectomy; Op. 2 = Right topectomy.

is in no way different from what could happen in non-addict patients submitted to topectomy. The most striking observation is Case 10, who was on more than 10 cc. of Demerol daily before the operation. This patient, who belongs to a high social group and is concerned with many business dealings, is now back at his former position with an entirely normal mental state.

This apparent cure of toxicomania, realized dramatically without any signs of withdrawal, has been observed before. In Scarff's article[9] on unilateral lobotomy, for instance, this was stressed, but morphine suppression was not always so immediate and so complete as in our own cases. It leads to two conclusions: first, the so-called "withdrawal accidents" are just secondary effects consecutive to an excessive anxious agitation; second, there are no profound physiological changes related to drug addiction, with the exception of the disturbance of the anxiety or *souffrance* cerebral circuit.

In the same line of physiological action of topectomy, occasional cure of impotence in male subjects was observed. In our Case 10, complete impotence existed for 6 months, and disappeared 3 weeks after the operation. Of course the mechanism of this is not quite clear; it may be due to the suppression of anxiety, or to the suppression of drug addiction, or both.

From a practical point of view, in patients having a mixture of organic unbearable pain and an abnormal mental state, no operation such as cordotomy or tractotomy should be tried. Bilateral lobotomy we disapprove of, as the potential risk of mental deterioration is too great. Bilateral topectomy is strongly advised, and unilateral lobotomy is a possibility if further observations confirm its value.

PHYSIOLOGY OF UNBEARABLE PAIN

Unbearable pain has been observed for many years, but little is known about its mechanism. The work of Weddell and his associates[12] is an important step in the sense that unpleasant pain seems related to an abnormal innervation of the skin (one terminal instead of several). If this is confirmed, it will give a satisfactory explanation for unbearable pain following all sorts of peripheral nerve lesions. In any event, it gives an anatomical basis to a special quality of pain.

Our work on topectomy may, it is hoped, belong to the same field. Unpleasant or unbearable pains of different origins are likely to express the disturbance of a single cerebral function. We call *souffrance* this disturbance, and in some cases, a way of measuring its variations is given (causalgias, thalamic syndromes). Furthermore, *souffrance* always showed a close parallel with anxiety. It is true that few physiological tests were used. We rely mostly on grosser impressions; the patient's personal feeling and behavior, the family's opinion, quality of social contacts and of professional work. All these things being considered, it can be said that the effect of topectomy on the mental action of these patients seems very much a specific effect on anxiety. This is only a confirmation of the very detailed studies of the Columbia-Greystone Group.[2] In no other mental activity were the patients disturbed; they resumed their work with the same knowledge and initiative as before; they seem to have the same drive in planning and trying to take care of business difficulties; they do not show any indifference to their affective life; if anything, they appear to be more inclined to social life than before.

Bilateral topectomy limited to the zone we term briefly "areas 9, 10 and 46" seems, therefore, to have a dual and limited effect: one on *souffrance*, one on anxiety. It is suggested that *souffrance* and anxiety are different ways of looking at the same cerebral function. Very little more can be said at the present time, except that this function is probably regulated through a circuit defined by at least three relays: dorsomedial nuclei of the thalamus, granular prefrontal cortex, and hypothalamus.

The anatomical and physiological proofs of such a circuit are progressively being given, thanks to the studies of Mettler,[7] Le Gros Clark,[1] Ward and McCulloch,[11] and Murphy and Gellhorn.[8] It is tempting to assert that the thalamo-prefrontal-hypothalamic circuit, when interrupted by topectomy, does not allow some or all modified spinothalamic impulses to reach the brain through mechanisms which play a primary role in the phenomenon

called *consciousness*. This of course goes much too far beyond actual experimental knowledge, and in that connection it is to be hoped that such a technique as thalamotomy (Wycis[10]) will be able to yield precise information in the near future.

Before closing remarks on the theoretical aspect of relations between topectomy and *souffrance*, the question of specific localization in the prefrontal cortex should be discussed. Is there a limited zone in the prefrontal cortex which is concerned with the development of unpleasant pain? Roughly speaking, two tendencies oppose each other, advocating either a qualitative effect or a quantitative one. It should be stressed again that the expression area (9, 10, 46) is a single topographical symbol, meaning little more than a certain part of the prefrontal granular cortex. Even so, partisans of the quantitative effect hold that the mental action of topectomy is proportional to the amount of resected cortex and does not depend on any particular prefrontal zone. The excellent psychological studies made on Pool's patients with mental disorders are not in favor of such a theory, since they showed a marked effect of ablation of 9, 10, 46 on anxiety. The study of our patients with pain, most of them mentally normal, is in favor of the theory of qualitative action. It seems a fact that there is a threshold in the necessary amount of excised cortex, but this is in no way a refutation of specificity. The main evidence against specific action comes from the good results of Scarff's unilateral lobotomy, as opposed to our own poor results with unilateral topectomy. The first step should be to confirm without any doubt this difference in action on unbearable pain, which has by no means been done as yet. Even so, only comparable patients should be used, for instance, patients in whom *souffrance* is objectively measurable (as in our causalgic or thalamic cases). In 1 case of thalamic syndrome, unilateral left lobotomy was not a success in that respect, and for the time being, it is our opinion that unilateral lobotomy brings about only a partial improvement of *souffrance*, as compared to bilateral topectomy. Further studies are necessary.

SUMMARY AND CONCLUSIONS

1. Following Pool's lead in the surgery of mental disorders, bilateral topectomy of the 9, 10, 46 region was performed since 1947 on 25 patients suffering from different kinds of unbearable pain.

2. Results are generally good with a bilateral cortical excision of a total of about 20 to 25 grams. Unilateral topectomy failed often. Some results have lasted nearly 2 years to date.

3. Different classes of unbearable pain are analyzed. The action of topectomy is beneficial on both organic unbearable pain or the so-called "psychic" pains. Indications for other operative procedures are discussed.

4. An objective technique of measuring unpleasant and unbearable pain is given. Such a condition is called *souffrance*, and its close relationship with

anxiety is shown. A step is taken in bringing a mental condition up (or down) to a physiological phenomenon.

5. Possible mechanisms and localizations involved are briefly discussed.

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TECHNICAL NOTE

A MODIFICATION OF THE McKENZIE SILVER CLIP

WILLIAM DUANE, JR., M.D.*

(Received for publication June 15, 1949)

In 1911 Harvey Cushing[1] introduced the silver clip as a means of hemostasis in neurological surgery. The original clip was made by winding silver wire in a tight coil around an oval or diamond-shaped rod and then cutting off the individual clips with a wire cutter. This made small U-shaped bits of wire which were then held in the jaws of an ordinary clamp for application to the blood vessels.

The author has never used the original Cushing clips, but according to McKenzie[2] the sides of the clips were often uneven in length, the ends rough, and the round wire caused a tendency for the clip to turn in the holder.

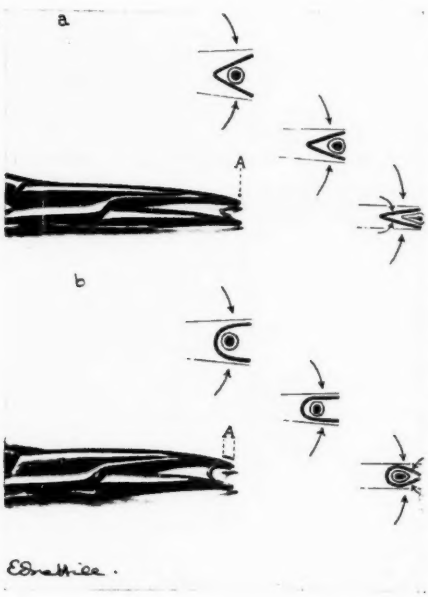

FIG. 1. Drawings comparing use of (a) V-shaped, and (b) U-shaped silver clips.

McKenzie in 1927 devised the instruments now in use: a punch which cuts out perfectly uniform V-shaped clips from a flat wire, and special clip holders for applying them.

It has been our experience that the V-shaped clips also have a tendency to wobble, and that they are easily dislodged when applying them since only the tips of the open end of the V come in contact with the grooves in the holder. Furthermore, we found that when applying a clip to a large blood vessel occasionally the vessel was squeezed out of the clip or only pinched by the open ends (Fig. 1, a).

To overcome this we had instruments made similar to those of McKenzie, but which punched out U-shaped, flat wire clips instead of the V forms. These were placed in an appropriately U-shaped magazine.

The clip holder is the same as McKenzie's except that it is made slightly heavier to prevent a tendency of the jaws to spring a little due to the necessity of a little more force needed in flattening out the clip.

In Fig. 1, b it will be seen that the parallel sides of the U-shaped clip give a longer area of firm contact in the grooves of the holder, thus preventing any turning or

* University of Pennsylvania, The School of Medicine, Philadelphia 4, Pennsylvania.

provocative dislodgment. Furthermore, as the clip is squeezed the arms of the U close, to encircle the vessel before compressive obliteration occurs, thus being more certain of accomplishing the desired effect. A crude example of the grasping properties of the two types of clips is in the approximation of one's index and middle fingers as compared to that of the thumb and index finger.

In conclusion, a U-shaped silver clip has several advantages over the V form in that the area of contact between the clip and the holder is greater, preventing to a greater degree the turning or dislodgment of the clip. Secondly, this clip when applied encircles the vessel before compression is completed, thus eliminating a certain tendency of large, sclerotic or elastic blood vessels to be forced out of the clip or torn by the tips of its sides.*

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* The instruments may be obtained from George P. Pilling & Son Co., 3451 Walnut Street, Philadelphia 4, Pennsylvania.

Obituary

COBB PILCHER

1904–1949

In the death of Cobb Pilcher neurological surgery has lost one of its most active and promising minds. For though he had made an amazing number of valuable contributions to the subject considering the short time he had been working in this field, it was quite clear to any observer that he had hardly reached his real stride. His work and industry gave promise of much greater things to come. He had a fertile mind and had that rare gift of selecting research problems that gave promise of yielding results.

His active life as a surgeon was divided between the problems of the care of neurosurgical patients and research that was suggested to him in the course of the care of such patients.

In going over his many publications, which numbered over sixty, it is quite evident that his interests were primarily in throwing light on factors that would improve our understanding of clinical disorders and thus helping in their treatment. This attitude was well illustrated by his series of papers on experimental cerebral trauma, the first of which appeared in 1937 and the last in 1941, and by his prolonged research on the role and action of chemotherapeutic agents in the treatment of infections of the nervous system. These investigations were carried on for the War Department during the recent war and helped to formulate rules in the treatment of intracranial infections. It was these studies that proved the danger of using sulfa drugs in cerebral wounds.

In the last three years he had been particularly interested in angiomatous lesions of the brain and his last publication was on this subject, presented before the International Neurological Congress in Paris one month before his death.

Cobb Pilcher was born in Nashville, Tennessee, on October 7, 1904, the son of M. B. and Mary Cobb Pilcher. He received his early schooling in Nashville and received his A.B. and M.D. degrees from Vanderbilt University. He was graduated at the early age of 22. Following this he interned at the Peter Bent Brigham Hospital under Harvey Cushing and then spent four years at Vanderbilt University Hospital as assistant physician, assistant resident surgeon and finally as resident surgeon. After this he worked with Percival Bailey in neuropathology in Chicago and with Ernest Sachs at Washington University, St. Louis, in neurological surgery. He then returned to Vanderbilt where he took charge of neurological surgery in 1934 and continued his activities up to the time of his death on September 22, 1949, at the age of 45.

During these years his time was taken up by a very active practice, research, and an engrossing teaching schedule. He was an inspiring teacher and imparted his enthusiasm to his students and aroused their interest to such an extent that a number of them were prompted to take up the study of neurological surgery. His interest in teaching will be continued at Vanderbilt by the creation of the Cobb Pilcher-William Henry Howe Fellowship in Neurosurgery.

He was a member of numerous medical societies, the most important of which were the Society of Neurological Surgeons, the Harvey Cushing Society, the American Surgical Association, the Southern Surgical Association and the In-

national Society of Surgery. He was president of the Harvey Cushing Society in 1948 and president of the Nashville Surgical Society, and chairman of the section on neurosurgery and psychiatry of the Southern Medical Association. It was while he was president of the Harvey Cushing Society that he delivered a memorable address entitled "Neurosurgery Comes of Age." This gives us the best picture of what his future career gave promise of and what a loss his early death has been to neurological surgery.

This brief picture of his work gives no indication whatever of his character and varied interests.

As a member of the Executive Faculty of the School of Medicine he proved himself very helpful. On matters of policy he was clear-cut in his convictions and vigorous in advocating them but always considerate and tolerant of other people's views. In recent years a number of important issues had to be considered by the faculty. In all discussions he was always concerned with one thing only: what was best for the School of Medicine. Once convinced of what was best he worked unremittingly towards its accomplishment.

He was a devoted, loyal friend and an entertaining, delightful companion. His many lay and professional friends bear witness to these qualities. He was a loyal Vanderbilt alumnus and showed his interest in the institution and in the students by innumerable kindnesses. His generosity towards students was one of the things that few even of his intimate friends knew anything about. Many a young student received advice and encouragement and financial aid from him and was started on his career through Dr. Pilcher's generosity.

He was so engrossed in his work and such an omniverous reader, that he had not had time to develop any hobbies, though he did enjoy fishing and the out-of-doors. What spare time he had he devoted to his wife and two lovely girls. In his home with the help of his wife he had created a beautiful atmosphere and he shared his hospitality with any friends or acquaintances who came to Nashville. His untimely end has deprived Vanderbilt University and neurological surgery of one of its most promising figures.

Ernest Sachs, M.D.

NOTICES

INSTITUTO CENTRAL DE NEUROCIRUGÍA Y NEUROPATOLOGÍA

On November 10, 1949 the Instituto Central de Neurocirugía y Neuropatología of Santiago, Chile celebrated its tenth anniversary. The most important event was the opening of two new departments: Neurological Surgery for Children, and Rehabilitation for Paraplegics. Many young physicians have come to the Institute from different countries of the Continent to avail themselves of the opportunities for specialization in neurosurgery. Professor Alfonso Asenjo, well-known Chilean neurosurgeon, is Director of the Institute.

THE NEUROSURGICAL SOCIETY OF AMERICA

The Neurosurgical Society of America was founded in Chicago, Illinois, on June 5, 1948 and is incorporated in the state of Illinois. The objects of the Society are the dissemination of information in the field of neurosurgery and the stimulation and promotion of fellowship among young neurosurgeons. Membership is open to neurosurgeons under the age of 45 who have completed the training requirements of the American Board of Neurological Surgery or its equivalent in the opinion of the Membership Committee of the Society. Application forms for membership may be obtained from the Secretary, Dr. C. D. Hawkes, 22 North Manassas Street, Memphis 5, Tennessee.

The first meeting of the Society was held in Chicago, November 19 to 20, 1948, and the second at Quebec, Canada, October 31 to November 2, 1949. The present membership of the Society numbers forty. The current officers are: President, Dr. Frank Otenasek, Baltimore; Vice-President, Dr. George Roulhac, St. Louis; Secretary, Dr. C. D. Hawkes, Memphis; and Treasurer, Dr. I. Joshua Speigel, Chicago.

SKIN RESISTANCE CHANGES IN THE LOWER LIMB AFTER LUMBAR GANGLIONECTOMY

A. HALL RATCLIFFE, D. PHIL. AND R. P. JEPSON, F.R.C.S.
Professorial Surgical Unit, Royal Infirmary, Manchester, England

(Received for publication June 3, 1949)

THE estimation of sweat gland thermo-regulatory activity has, in the past 20 years, been increasingly utilised, as an objective method of localising and interpreting lesions in the central, peripheral and sympathetic nervous systems. In many cases, such as those of lesions interrupting and limited to the sympathetic pathways, the detailed plotting of anhidrotic skin is the only clinical method available that can accurately outline the cutaneous area affected by the neural section. The revelation of such anhidrotic areas may therefore be used, diagnostically in those rare selective types of trauma, the sympathetic chain stretch injuries described by Guttmann,[4] or more commonly, as a means of evaluating the effectiveness and extent of surgical sympathectomies. It is with the disturbances of the thermo-regulatory sweat gland activity in the first 10 days after lumbar ganglionectomy that this paper deals. A new technique for measuring skin resistance changes is also described.

For the measurement of thermo-regulatory sweating in both clinical and physiological practice, two entirely different methods have been developed, the "chemical" and the "electrical."

The former depend on the colour changes resulting from the interaction of the skin surface moisture and a colorimetric substance which has previously been applied to the skin. The original substance used was starch and iodine.[8] This has largely been replaced as a colour indicator by the red-brown dye quinizarin (2-6-di-sulphonic acid).[5] The chemical methods unfortunately have many disadvantages. They all necessitate the heating of the patient sometimes to an uncomfortable degree, in order to exaggerate the contrast between the sweating and non-sweating areas, and in the constitutionally hypohidrotic person, syncope may result before an adequate pattern can be demonstrated. It is not possible therefore to examine patients satisfactorily by this technique within a few days of a major operation. The heating is most suitably done in a special and rather cumbersome sweat cabinet,[5] as reflex thermo-regulatory sweating by immersion of the limbs in hot water will often not reveal the finer details. Heating by ordinary electric lamp cradles is unsatisfactory and may result in burns, especially over anaesthetic areas. The quinizarin powder may be difficult to apply evenly on shiny skin and is always unpleasantly dirty to the patient especially around the head and neck regions, where sneezing or lacrimation from powder irritation may also invalidate the findings. Finally, the interpretation of the resulting sweat patterns requires meticulous attention to detail and considerable experience

in the method. Absolute measurements of sweat gland activity cannot of course be made by the chemical techniques.

The electrical methods are based on the finding that the electrical resistance between two skin electrodes varies with the amount of sweat gland activity occurring in the skin under the electrodes.[2,10,12,13,14,15,19,20,21,23] The actual distance between the skin electrodes is of little account.

The electrical circuit between the electrodes A and B in contact with the skin may be represented in simplified form by the resistance network shown in Fig. 1. Here R_1 and R_4 represent the contact resistance between the electrodes and the tissue, R_2 represents the resistance between the electrodes at the surface of the skin, and R_3 represents the resistance through the tissues and appears in parallel with R_2. R_3 is small compared with the other resistances and virtually short-circuits R_2. Thus the reading obtained depends on R_1+R_4, that is, the contact resistance between the electrodes and the skin. This can be demonstrated by a simple experiment.

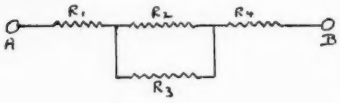

FIG. 1.

A subject was chosen whose skin was rather more moist than the average. A disc-electrode $\frac{1}{2}$ inch in diameter was strapped on the forearm just below the elbow, electrical contact being made to the skin through electrode cream. Direct contact with the skin was made at points 1, 2, 3, 4 and 5 inches away from the first electrode through a $\frac{1}{4}$ inch diameter spring-loaded electrode. The resistance between the electrodes was measured by means of a battery and micro-ammeter.

The values obtained in each case lay between 270,000 and 280,000 ohms, which may be regarded as constant within the limits of experimental error, or, in other words, the resistance is not dependent on the distance between the electrodes. Because the thermo-regulatory activity of the sweat glands is dependent on the integrity of their "sympathetic" sudomotor fibres, it follows that "sympathectomised" skin shows a higher electrical skin resistance than normally innervated skin. At a room temperature at 18°C. the difference between the resistance of normal and sympathectomised skin of the average patient is small, the resistance of the sympathectomised being of the order of 10 per cent higher than that of the contiguous normal areas. In the method developed by Richter,[11] and later modified by Jasper,[6] and Whelan and Richter,[23] the skin resistance is measured by putting the patient in series with a low voltage ($4\frac{1}{2}$–9 volts) battery and a micro-ammeter with a full-scale deflection of 20 microamperes. The indifferent electrode was originally clipped to the lobe of an ear, electrical contact being made with an electrolytic cream; later a simple zinc surface electrode was used. The exploratory electrode consisted of a small plated phosphor-bronze disc attached to a wooden handle. As the resistance over the normal skin was too high to give a meter deflection of enough magnitude, the patient was first heated in a hot-air cabinet until the resistance was lowered sufficiently by

sweating to give an adequate deflection. Then the exploratory electrode was tracked over the patient and when the meter reading fell, owing to contact with high resistance skin, this was assumed to be the "level" of the sympathectomised area.

The advantage of the above direct system of resistance readings over the quinizarin sweat test, is its speed and simplicity of usage but unfortunately it fails to eliminate, because of its insufficient sensitivity, some preliminary heating of the patient. Direct stimulation of sweat gland activity by pilocarpine, mecholyl or furmethide is fallacious. This necessity for heating the patient can be overcome in part by increasing the delicacy of the recording apparatus at the expense of its robustness, or by increasing the amount of current passed through the patient. The latter results in polarization–invalidating the readings—or in unpleasant subjective side-effects to the patient. To obviate this troublesome necessity of heating the patient before testing we have developed the following technique.

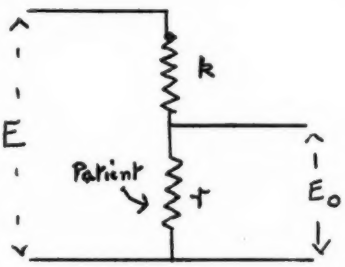

FIG. 2.

For the charting of sympathectomised skin it is not necessary to measure its absolute resistance but merely to demonstrate adequately the boundary zone between it and the contiguous normal skin.

If the patient is made the lower limb of a potential divider circuit as shown in Fig. 2, the output voltage E_0 will have the relationship to the input voltage, E, expressed by the equation:

$$E_0 = \frac{r}{k + r} E.$$

This means that the output voltage depends on the patient's skin resistance and will increase with increase of this resistance. Instead of employing a battery for the source of input voltage, E, a pulse generator was used. This has two marked advantages, first that subsequent amplification can be accomplished without the necessity for complicated D.C. amplifier systems and secondly that a constant amplitude pulse can be produced relatively easily. The voltage produced across the patient's resistance may be shown diagrammatically in Fig. 3a for "dry" sympathectomised skin and in Fig. 3b for "moist" normal skin.

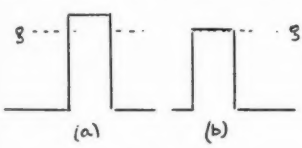

FIG. 3.

If the pulse produced as E_0 is passed through an amplitude "gate" circuit in which the gate voltage is set at the level shown by the dotted line g–g in Fig. 3a and b, only pulses of greater amplitude than this voltage

will get through the gate. The output from the gate circuit is fed to an amplifier system. After amplification the pulses from the dry and moist areas can be represented diagrammatically by Fig. 4a and b.

The amplitude of the resultant pulses may be evaluated by a peak voltmeter. By choosing suitable circuit constants it can be arranged that the meter reads zero over normal skin and almost full scale over sympathectomised skin. Full circuit and technical data of this electrodermatohmmeter (E.D.O.) will be published later.

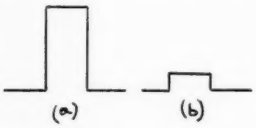

FIG. 4.

The electrode was made from a miniature bayonet socket of the type used for indicator lamps. The outer tube with the bayonet locating slots was cut down to a level which left the contact pins protruding for approximately 1/16 inch. The springs on the contact pins were lightened.

For the clinical application of the E.D.O. apparatus, the patient should preferably be tested in a thermostatically controlled room, though many of our early postoperative readings have been done in the ward so that the patient should be disturbed as little as possible. A room temperature of 20–21°C. is ideal although in plotting those patients with constitutionally dry skins a few degrees higher than this is advantageous. The area to be investigated is uncovered and left exposed to the room temperature in order to eliminate contact moisture. The electrode is then placed on non-sympathectomised skin—the abdomen in lumbar ganglionectomies—and the circuit adjusted until a zero (no deflection) reading is obtained. Skin readings are then made axially along the limb until the demarcation line between "dry" and "moist" skin is found. This is shown by the needle, which on sympathectomised skin approaches full scale deflection, returning rapidly to the zero position as normal skin is reached. When the rough level is ascertained the whole demarcation zone can be accurately determined. With a little practice and experience, a lumbar ganglionectomy pattern can be "mapped out" in a few minutes.

MATERIAL INVESTIGATED

Twenty-seven patients were investigated; of these 15 had had bilateral and 12 unilateral lumbar ganglionectomies. A total, therefore, of 42 postoperative limb patterns was studied. Table 1 shows the complaints for which the operation was performed and also the sex distribution of the patients.

All the lumbar ganglionectomies were performed by the same surgeon, using an extraperitoneal oblique flank approach. With the exception of the hypertensives (8 cases) and the 1 case of Hirschsprung's disease, the resected lumbar sympathetic chain was that readily available below the diaphragm and was limited to a segment 2–3″ long opposite the body of L3 vertebra.

As Telford[18] has pointed out, "the resection of the trunk cannot be described in terms of ganglia but is best defined as resection of 3–5 cm. centered

opposite the 3rd lumbar vertebra." This usually involved the removal of two ganglia and intervening chain as judged by the gross anatomy and some were checked by further histological section. It is probable that the ganglia removed by this operation supplied grey rami communicantes to the L2, 3, 4 nerve roots.[1] It is unlikely that the highest lumbar ganglion is removed by this technique as it lies high under the crus of the diaphragm, which must be split by the surgeon for its exposure. The lowest lumbar ganglion (or ganglia) hidden behind the common iliac arteries was not searched for. In the hyper-

TABLE 1

	No.	Female	Male
Raynaud's syndrome	4	4	—
Embolism (femoral)	1	1	—
Hypertension	8	5	3
Thrombo-angiitis obliterans	4	1	3
Hirschsprung's disease	1	—	1
Obliterative vascular disease	7	1	6
Venous thrombosis	2	2	—

tensive group, plus the 1 case of Hirschsprung's disease, a careful dissection was made cephalically, splitting the crus of the diaphragm to include the highest lumbar ganglion in the resection, and occasionally the lowest thoracic when this was present.

FINDINGS

All the findings reported here were made within the first 10 days post-operatively; each case was usually investigated many times on different days within this period. The level of readings on any patient were constant during the period. This is contrary to the findings of Ray and Console,[9] who found the level migrated distally during the immediate postoperative period but seems to be in accordance with most other workers in this field.

The demarcation zone between sympathectomised and normal skin is usually extremely sharp. Occasionally this is not so, and a zone of "intermediate moisture" with a breadth of a few cm. separates the two. More rarely the division is marked by a hyperhidrotic fringe—the so-called "perilesionary" hyperhidrosis.[5] Neither of these variants, when recognised, adds to the difficulty of the investigation.

Within the high resistance area of sympathectomised skin, "oases" of low resistance may survive. The largest detected was about the size of the palm of the hand. They appear to be temporary phenomena and usually disappear within a few days. It is usual for the foot over a "slipper" area (Fig. 5), including the toes, sole, instep, to present immediately postoperatively a low resistance, about equal to that of normal trunk skin at room temperature. This contrasts with the statement of Brown and Adson[2] that "the driest areas occurred in the feet." As the affected foot is warm to the limit of maximal vasodilatation, of pink colour, and the limb shows a "resist-

ance level," it may be assumed that the leg has nevertheless been adequately sympathectomised. These "slipper" areas gradually contract in size, disappear after a period of months. We have not seen in our series the onset of gross sweating, colour changes, coolness of the lower (in contrast to the upper) extremity, occurring 3–6 days postoperatively, as described by Smithwick,[17] and White and Smithwick.[24] That the slipper area is due to spontaneous activity arising in the decentralised sympathetic ganglia would seem unlikely in view of the persistence of this low-resistance phenomenon for many months.

The actual level of skin resistance change varied considerably in the series. Of the 42 limbs examined, 25 were what we have designated a "high" level. A typical example is shown in Fig. 5. The line is sharp and smooth,

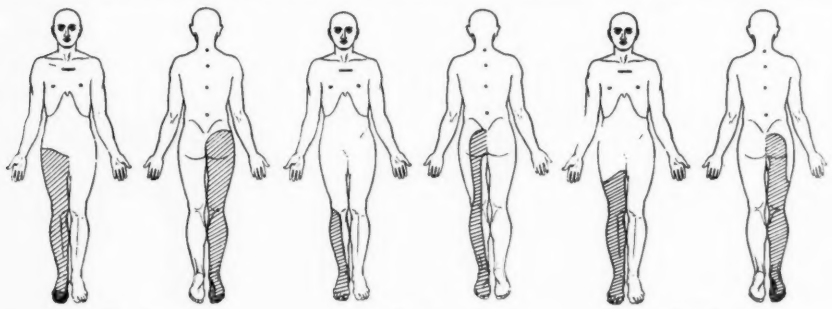

FIG. 5. FIG. 6. FIG. 7.

with no marked indentations or encroachment, running anteriorly just below Poupart's ligament to the suprapubic region—where it is lost amongst the hair—and posteriorly about the crest of the ilium to the midline. There is a very sharp line of demarcation at the midline with no overlap on either side. There were minor variations in the group but all were essentially this standard pattern when assessment had been made for the anatomical configuration of the patient. All the cases of hypertension (12 limbs) in which, as previously stated, a determined effort had been made at operation to remove the lumbar chain as high as possible after splitting the crus of the diaphragm were included in this group. The remaining 13 limbs had been sympathectomised for conditions other than hypertension and in these the "limited" operation of removing 2″–3″ of the chain opposite L3 vertebra had been performed.

In 7 limbs a "low" level was found. Anteriorly the demarcation line ran roughly along the crest of the tibia to below the internal malleolus inferiorly and round the head of the fibula superiorly. On the posterior aspect of the limb these continued upwards to the middle of the sacrum including the posterior lateral aspect of the calf, a strip of the thigh and a disc of buttock in the anhidrotic area (Fig. 6). In all the "low level" group the "limited" operation had been performed.

Finally out of the 42 limbs, 10 were in an intermediate group. The anterior level was not so constant as in the high and low level groups, but fell somewhere on the lower two thirds of the thigh. The whole of the calf and a variable strip of the posterior thigh and buttock were included in the anhidrotic area. Fig. 7 is an example. All the 10 had had the "limited" procedure.

DISCUSSION

To deduct the anatomy of the lumbar ganglionated chain from a basis of cutaneous sweat changes is in our view both fallacious and profitless. The anatomy of the chain is extremely variable,[1] and the distribution of the grey rami bewildering in its complexity. Its diameter may vary from a fraction to several mm., it may be single or duplicated, the number and position of its ganglia are variable and their connections are notoriously inconstant. Occasionally the whole chain may be a diffusely ganglionated mass, without visible intervening thinning. It is justifiable to infer, however, from our findings that in those ganglionectomies in which an effort is made to extend the resection up behind the crus of the diaphragm, the whole of the lower limb will show skin resistance change and may be presumed to be completely sympathectomised, whilst in those in which a segment of the chain opposite L3 vertebral body is removed a variable pattern, unpredictable within wide limits, will be encountered. This accords with the reports of Smithwick,[16] Richter and Woodruff,[15] List and Peet,[7] Fontaine, Houot and dos Santos,[5] who consider that the highest lumbar ganglion should be removed if it is wished to denervate the upper thigh area. In striking contrast is the statement of Ray and Console,[9] "Complete denervation of the lower extremity, therefore, is not determined by preservation or removal of L1 ganglion or any part of the paravertebral ganglionated chain above it." Ulmer and Mayfield,[22] have gone so far as to suggest that the removal of the lower thoracic ganglia may be necessary in addition to the resection of the lumbar chain, in order to guarantee a high thigh denervation. With this we would most strongly disagree. In those hypertensive patients who have had the thoracic sympathetic chain removed following a previous high lumbar ganglionectomy, no increase in the area of high electrical skin resistance has been found.

This question of high thigh denervation is not a matter of purely theoretical importance. For in those cases of arterial disease in which the thrombus extends into the common femoral region, it is imperative to ensure the maximal collateral dilatation in the region above the block. This can be obtained, it would seem, only by including the retrocrural sympathetic chain in the resection. A sympathectomy that dilates only the collateral circulation distal to the arterial block may be causing a proportionately greater shunting of the limited blood supply through the skin of the distal limb, and thus impair rather than benefit the general limb nutrition. We therefore feel from the evidence available both in the literature and from our own findings that in cases of arterial disease involving the thigh a "lum-

bar" sympathectomy should include the highest lumbar ganglion, which can only be regularly performed after the splitting of the diaphragmatic crus. It should be stressed that in the whole of the limbs examined in this series the foot has been sympathetically denervated as shown by colour and temperature change. The levels found by our technique in this series have been checked in many cases by the more established methods of Richter and by the quinizarin sweat test and similar results obtained, although the "oases" and "slipper" areas were not adequately demonstrated.

SUMMARY

1. Methods of detecting sudomotor fibre interruption are discussed.

2. A new technique for the investigation of sympathectomised skin is described.

3. Patterns of high skin resistance after 42 lumbar ganglionectomies are described. It is shown that the highest lumbar ganglion must be removed if the upper thigh is to be surely denervated.

Our thanks are due to Professor A. M. Boyd under whose guidance this investigation was pursued, and to Miss D. Davidson for the illustrations.

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THE MECHANISM OF SKULL FRACTURE

E. S. GURDJIAN, M.D., JOHN E. WEBSTER, M.D., AND H. R. LISSNER, M.D.
Department of Surgery, Wayne University, and Department of Neurosurgery, Grace Hospital, Detroit, Michigan

(Received for publication June 4, 1949)

UNTIL recently, the mechanism of the production of linear skull fracture was not accurately understood since the over-all deformation patterns of the skull due to a blow were not known. Previous investigators have stated that a local deformation results in a depressed fracture and that general deformations result in linear fractures. This concept cannot be verified by a study of the actual deformation patterns. All impacts upon the skull result in local and general deformations of varying intensity if the energy expenditure is adequate. The production of a depressed fracture depends mainly upon the velocity of the injuring object, and to a less extent, its size and shape. A single linear fracture will result if the velocity of the blow is below a critical value and the energy available is between certain limits.

Aran,[1] in 1844, described his irradiation theory of fractures. He stated that a fracture of the vault from impact on the vertex reached the base through the shortest possible route, implying that fracture started at the point of impact. Félizet,[3] in 1873, stated that fractures result when an impact flattens out the curved surface of the skull. The fracture may then be guided by the presence of buttresses. He also observed that separation of buttresses, one from the other, due to an impact, may result in a fracture between them. His classical description of the buttresses of the skull is frequently quoted. He described single mid-frontal and mid-occipital and paired fronto-sphenoid and parieto-petrous buttresses. Bruns,[2] in 1854, working with static loads found that compression of the skull resulting in shortening of the diameter in one direction, produced an increase in the diameter in the direction at right angles with the former. He also noted that although there was a certain degree of elasticity, the skull never returned to its former shape following static loading. He showed that the skull might be compressed from side to side, resulting in a shortening of its side-to-side diameter by as much as 12 to 14 mm. without failure. He found that when the load was removed, the side-to-side diameter was 4 to 5 mm. less than before its application. It was Bruns' theory that pressure applied along one diameter would result in tearing apart forces in the portions of the skull at right angles to the direction of the force. In other words, that part of the skull where the radius of curvature decreased or where outbending took place eventually cracked. Obviously, compression when carried too far also resulted in fractures and depression at the point of application of the force. Rawling,[6] in 1905, described his concepts of the mechanism of fractures involving the base of the skull. He stated that fractures of the base result from the force

of the blow actually splitting the skull in the same manner that a hatchet can split a piece of board along its grain. This again implied that the fracture started at the point of impact. The work of LeCount and Apfelbach,[5] in 1920, is also interesting in that they more or less substantiated some of the former concepts enunciated by Aran, Félizet, Bruns, and Rawling. In a recent text on head injury by Rowbotham,[7] essentially the above material is given for the mechanism of skull fracture. For instance, the author speaks of local deformations resulting in depressed fractures and generalized deformations producing linear fractures.

In the next few paragraphs it will be shown that all blows to the head result in local deformations as well as general deformations. In the production of a linear fracture, the local deformation from an impact is elastic, the bone rebounding after the blow. In the depressed fracture, the bone fails locally at or about the point of impact.

CERTAIN ANATOMICAL CONSIDERATIONS OF THE SKULL

The skull is made up of vault and base. The vertex through its greatest extent consists of two layers of bone with an intervening cancellous structure constituting the diploë. Certain anatomical variations may occur in the vertex. Although most frequently the outer table is thicker than the inner table, at times the inner table may be quite thick due to the presence of certain diploic ramifications through the outer table of the skull. These variations in the anatomy of the flat bones of the skull may be important in the resultant deformation patterns, particularly as concerns fracture of the inner table. The base of the skull consists of membranous bone and it appears to be extremely brittle under impact. Many foramina are found at the base and these are areas of stress concentration and fractures usually extend toward foramina. Battle, Rawling and others recognized foramina as regions of weakness at the base.

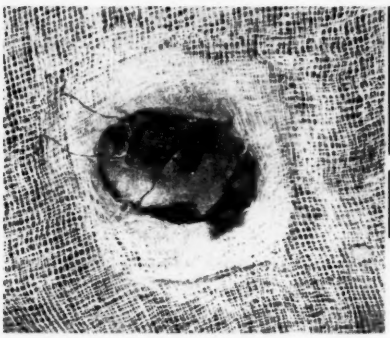
Most injuries to the skull are due to blows over the vertex and along the junction of the vertex with the base. A few injuries undoubtedly result from impacts so delivered as to cause the spinal column to extend into the cranial cavity. Injuries may result from blows toward the base, such as a blow on the lower jaw, thrusting the condyloid processes of the jaw against the base of the skull. Occasionally, the head may be compressed between objects (such as a car and the ground).

OBSERVATIONS

Time Period of Disturbance Following Blunt Impact. With the use of modern techniques, it has been possible to determine the time period of disturbance following impact upon the human head and to study the deformation patterns that result from impact. The total time period of disturbance following impact is in the neighborhood of 1/250 of a second.*

* This work was described in the paper "The mechanism and management of injuries of the head," by E. S. Gurdjian and J. E. Webster, *J. Amer. med. Ass.*, 1947, *134:* 1072–1076, and is based upon strain gauge studies of deformations of the skull in human autopsy heads.

During the first 0.0006 of a second following the impact, the scalp compresses. At this point the deformation of the skull begins. The deformation of the skull resulting in fracture takes another 0.0006 of a second. In Fig. 1, a strain gauge record of a cadaver head with scalp and contents intact is shown. Following a deceleration impact, the instant of contact of the scalp is noted by the disappearance of one line of a two-gun oscilloscope tube. The other channel is used to show the deformation of the skull proper. In this particular specimen, the fracture line crossed the strain gauge, thus completely opening the circuit. The time at which the circuit opened may be considered the time of initiation of the fracture line. From the time of



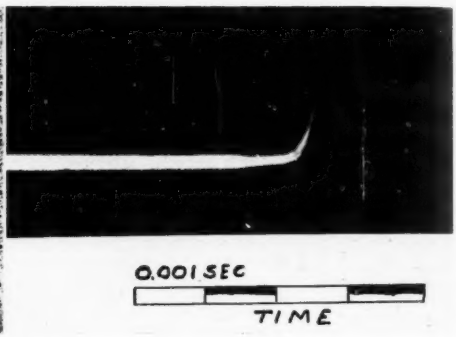


FIG. 1 (*left*). Human cadaver head with scalp and contents intact prepared for strain gauge measurement. The portion of the scalp and subcutaneous tissue has been removed from over the skull and the strain gauge has been placed in position with glue. Following the deceleration impact the fracture line completely disrupted the strain gauge. The oscillographic record of this case may be seen in Fig. 2.

FIG. 2 (*right*). A two-gun oscillographic record with one beam to show the time of contact of scalp with steel slab and the second beam of light to show the deformation of the skull ending in a fracture which opened the circuit. It is to be noted that there elapsed 0.0006 of a second from the time of contact of the scalp with the steel slab to the time of beginning deformation of the skull and there elapsed another 0.0006 second until the bone fractured and the circuit was opened since the fracture went right through the strain gauge. (Retouched)

contact of the scalp with the steel anvil, to the time of beginning of deformation of the skull, 0.0006 second elapsed. From the time of beginning of deformation of the skull, to the time when the circuit was opened (when the fracture occurred), another 0.0006 second elapsed. These figures may be of importance in connection with engineering techniques to decrease the rate of deceleration of the skull to counteract the injurious effects of blows of this magnitude and velocity occurring in auto and plane accidents.

Deformation Patterns Following Blunt Impact. Deformation patterns following impact were studied with the "stresscoat." This technique utilizes a strain sensitive lacquer applied both to the external and internal surfaces of the skull. The lacquer cracks due to tension stresses. The cracks in the lacquer appearing on the outside of the skull result from outbending. Those

appearing on the inside of the skull are due to inbending.* By this technique an accurate determination of the deformation pattern of the skull following impact may be obtained.

"Stresscoat" experiments have shown that following impact there is always an area of inbending immediately beneath and around the point of the blow. If the energy is adequate and if the velocity is sufficiently high, this area of inbending may fail, resulting in a depressed fracture. If the inbending is not severe enough to cause a fracture at the boundary of the inbended area, the skull rebounds. The inbending about the point of the blow is associated with discrete areas of outbending peripheral to the area

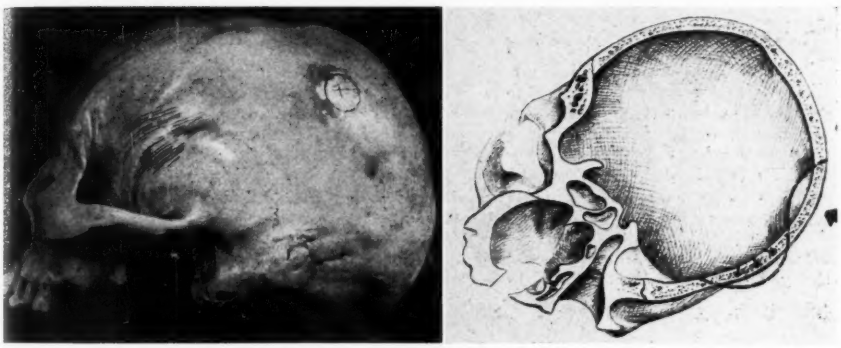


FIG. 3 (*left*). Posterior parietal blow in a freshly dried cadaver skull with resultant "stresscoat" pattern in the temporal region. Cracks in the lacquer suggest tearing apart forces from outbending in the temporal region from the blow in the posterior parietal. Immediately around the point of blow, one would find radially arranged cracks in the lacquer on the interior of the skull denoting the extent of the inbending from the blow.

FIG. 4 (*right*). Diagrammatic representation of the inbending-outbending following an impact in the posterior parietal region. If the area of inbending rebounds, the fracture initiated in the temporal region from outbending of the bone in that area extends toward the point of impact and in the opposite direction.

of inbending. This outbending may be severe enough to cause tearing apart forces resulting in a linear fracture. The fracture line then extends both toward the point of impact and in the opposite direction. It extends toward the point of impact rather than to one or the other side for the following reason: Initially the area of impact is inbended or in compression on the external surface, but immediately afterward, it is in tension or outbended. As such, it represents an area of tensile stress. The fracture line at a distance will be directed to this area of stress concentration since the maximum tensile stress will occur at the point of impact after rebound.

* Experimental work to show that it is proper to use the freshly dry human skull for study of deformation patterns may be found in "Deformation of the skull in head injury," by E. S. Gurdjian and H. R. Lissner, *Surg. Gynec. Obstet.*, 1945, *81:* 679–687.

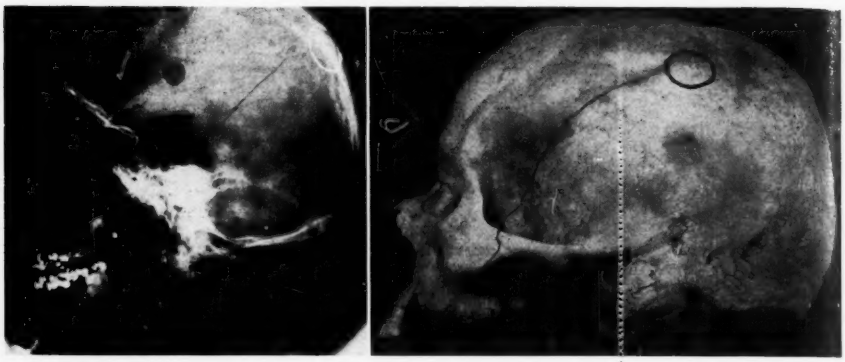


FIG. 5 (*left*). This patient fell sustaining a bruise in the posterior parietal region on the left side. The area of the bruise was considered to be the point of impact and circular radiopaque wire was placed surrounding this area. Note that the fracture extends toward this point from the temporal region. Note, also, that the fracture is much more evident in the temporal region than near the point of impact where it tapers to a fine line. The fracture began or was initiated away from the point of impact, and due to outbending of the skull. It then extended toward the point of impact because, although initially the area of impact was pushed in or in compression, immediately afterward, it rebounded and therefore it was in tension, and as such, a fracture would tend to extend toward this area with the bone in tension.

FIG. 6 (*right*). Cadaver head with scalp and contents intact following a posterior parietal blow on the left side with resultant linear fracture extending up toward the point of impact. Note that in this instance the zygomatic arch is also fractured.

FIG. 7 (*left*). "Stresscoat" preparation following a posterior parietal blow which was much heavier than the blow in Fig. 3. Note that cracks in the lacquer appear not only in the temporal region, but also in the parietal region superiorly and in the mastoido-parietal region posteriorly and inferiorly. The cracks in the temporal region represent the area of greatest weakness and if a single linear fracture were to result from a blow, the fracture would be initiated in this region; but if the blow were more severe, a second line of fracture would be initiated in the superior parietal region extending toward the point of impact and if the blow were still more severe, a stellate fracture would be obtained.

FIG. 8 (*right*). Diagrammatic representation of areas of stress level following a posterior parietal impact. The temporal area is the weakest area with tearing apart forces denoting the area of primary stress level (1). The superior parietal region is the next weakest region, denoting the area of secondary stress level (2), and the parieto-mastoid region inferiorly is the region of tertiary stress level (3). With adequate energy a single linear fracture would be initiated in the area of primary stress level. If still more energy were used, a second line of fracture would be initiated in the area of secondary stress level and if still more energy were utilized, then a stellate fracture would result.

With the use of the "stresscoat" it has been possible to delineate the areas of outbending following impact in a given region of the skull. It is interesting to note that as the energy of impact is increased, outbending is noted in more than one region. The location of cracks in the lacquer caused by outbending obtained with the smallest amount of energy expenditure represents the area of greatest weakness. This has been previously termed "the area of primary stress level." After energy expenditure of larger

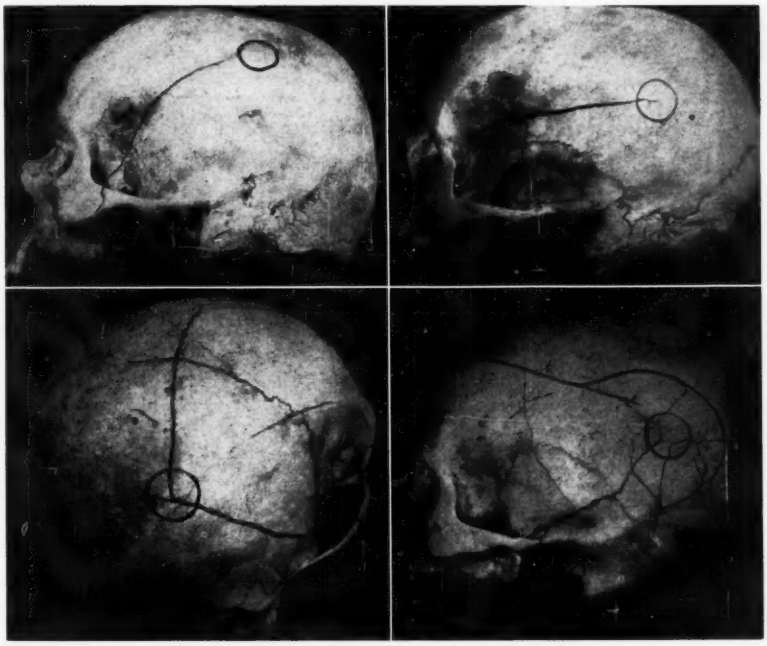


FIG. 9. These 4 skulls are from cadaver head experiments with scalp and contents intact. In the upper left, a single linear fracture is obtained from a posterior parietal blow. In the upper right, a two-line fracture is obtained from a posterior parietal blow on the left side. In the lower left, a three-line fracture is obtained from a posterior parietal blow on the right side. In the lower right, a stellate fracture is obtained from a posterior parietal blow on the left side. The resultant fractures are found to follow the "stresscoat" predictions quite accurately.

magnitude, evidences of outbending occur in a second area in addition to the area of primary stress level. This region is called the area of secondary stress level. With the expenditure of still greater amounts of energy, cracks are noted in the lacquer due to outbending in additional regions and these are called areas of tertiary stress level.

The significance of these results which have been corroborated experimentally by tests of intact cadaver heads is that the area of primary stress level denotes the region of greatest weakness, and where a linear fracture would be initiated with adequate energy expenditure. If more energy were

used to obtain more than a single linear fracture, the second line of fracture would occur in the area of secondary stress level, and if still more energy were used, a stellate type fracture would result.[4]

On the basis of "stresscoat" experiments upon 100 skulls, we have delineated the area of primary stress level following impact in 12 regions into which the skull surface has been divided. Observations show that an adequate blow in a given region generally results in a linear fracture in a specific area. If

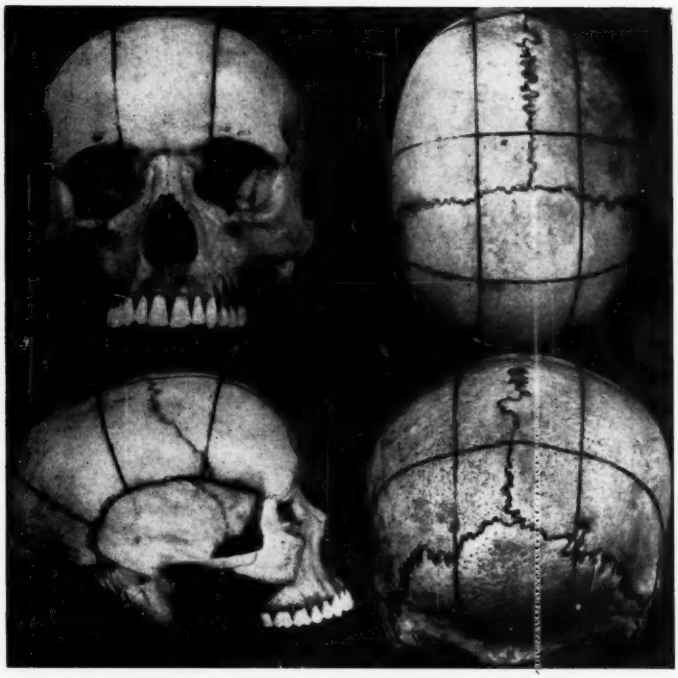


FIG. 10. The 12 areas into which the skull surface has been divided. Blows administered in each one of these 12 areas in 62 human skulls to delineate the area of primary stress level have been studied.

the area of impact is known, a prediction of the location of the fracture can be made with a reasonable degree of accuracy. By the same token, if the position of a linear fracture is known, the point of impact may be determined.

The velocity of the injuring object, provided the energy is adequate, is the important factor in the causation of depression. The higher the velocity, the greater is the possibility of depression and perforation. If the velocity is higher than a certain critical value, a neat hole is produced by the injuring object. Under these circumstances, distant deformations are minimal or absent. If the velocity is extremely high, the radial acceleration imparted to the skull by the missile causes extensive shattering. With the velocity of the blow lower than a certain critical value, varying degrees of dis-

tant deformations may be obtained with resultant linear fracture or fractures from outbending of the skull, accompanying the depression. Also, with higher velocity and energy, the area of inbending resulting in a depression may be more and more patterned after the shape of the injuring object. A bullet of average velocity may make a neat hole in the skull, whereas a baseball, which travels much more slowly, results in a rather large area of depression, with the bone fragmented by three to five lines arranged in a stellate fashion. The radius of curvature of the skull is another factor affecting the extent of the area of depression, in these lower velocity injuries.

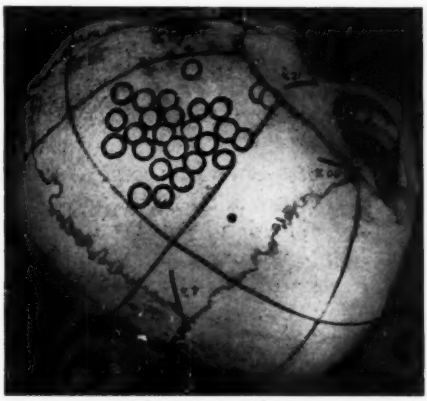


FIG. 11. In 25 examples of blows in the posterior parietal region on the left side, it was found that the area of primary stress level was in the temporal region in 80 per cent of the cases, in 12 per cent of the cases it was in the posterior temporal region just above the ear canal, in 4 per cent in the superior parietal area, and in the other 4 per cent in the parieto-mastoid region.

Mechanism of Depressed Fractures. There are six varieties of depression based upon the velocity, the kinetic energy and, to a lesser extent, the shape of the injuring object.

1. An object moving with extremely high velocity (a high-powered rifle bullet) will not only perforate the skull, but will cause shattering of the bone from radial acceleration imparted to the skull and contents, resulting in tremendous increase in intracranial pressure (bursting fractures).

2. An object moving with fairly high velocity (a pistol bullet) will perforate the skull resulting in fragments of bone being indriven into the substance of the brain. If the kinetic energy is not high and most of it is dissipated at impact with the bone, the outer table alone may be depressed or perforated.

3. A blunt object moving at a lower velocity than those previously mentioned, such as a baseball, brick or hammer, may cause an area of depression in which most of the expended energy is absorbed in producing the depression. Under these circumstances there is inbending of an oval or circular area of bone with fragmentation by three to six radial fractures and separation of the inner and outer tables. The border of the oval or circular area usually presents a curvilinear fracture and this is caused by tearing apart forces on the external surface of the skull at the junction of the inbended and not-inbended bone. The fragmentation of the area of depression roughly follows the pattern shown by the "stresscoat" on the internal surface of the skull around the area of impact and is due to tearing apart forces from inbending. The outer and inner tables of the skull may be torn apart. Under

the circumstance that the energy of the missile is almost completely dissipated at impact, the outer table may be depressed or if the inner table is thicker, the inner table may be depressed.

4. A slowly moving object, causing a localized blow upon the skull, may result in a depression with simultaneous deformation in regions other than the area of impact. Under these circumstances, not only is there an area of depression, but there are also one or two linear fractures extending toward the area of impact which have resulted from tearing apart forces from distant outbending of the skull.

5. A slowly moving object, fairly sharp or pointed in contour, may cause an area of depression more or less patterned after the shape of the object. Under these circumstances, distant deformations of the skull resulting in linear fracture or fractures may or may not be present.

6. Depression by a slow-moving, high kinetic energy blunt surface may result in extensive comminution with radial fracture lines extending from the center of impact and circular fracture lines at varying distances surrounding the area of impact. This type is frequently seen with deceleration impact.

CONCLUSIONS

The mechanism of skull fracture has been described. This work has been based upon "stresscoat" studies describing the deformation pattern of the skull following impact. Whether a depressed fracture or a linear fracture is obtained depends entirely upon the velocity, the kinetic energy and, to a less extent, the shape of the injuring object. The area of impact is inbended, and in discrete regions of the skull there are evidences of outbending if the energy expenditure is adequate. If the area of inbending following impact rebounds without fracturing locally, a linear fracture may be initiated at a distance from the area of inbending due to tearing apart forces from outbending of the skull. The fracture then extends both toward the point of impact and in the opposite direction. If the area of inbending fails due to the magnitude and velocity of the blow, a depressed fracture is obtained. Linear fracture or fractures from outbending at a distance from the area of impact may be obtained if the velocity of the injuring object causing the depression is not too high. Tearing apart forces due to an impact are produced by bending of the bone and not by direct stress.

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BILATERAL TORKILDSEN PROCEDURE

ITS APPLICATION IN INSTANCES OF OCCLUSION OF BOTH FORAMINA OF MONRO*

HOMER S. SWANSON, M.D., AND GEORGE PERRET, M.D.†
Department of Surgery, Emory University School of Medicine, Atlanta, Georgia

(Received for publication June 11, 1949)

THE operative procedure ventriculocisternostomy, first proposed by Torkildsen,[2] has become an accepted and effective method in relieving a non-communicating hydrocephalus. In a previous communication, presented before this group in 1947, our[1] accumulated experiences were set forth in utilizing this procedure. Since this report the operation has been performed in an additional 16 cases. It is the purpose of this discussion to report 3 instances in which a double drainage of the lateral ventricles seemed preferable to a single ventriculocisternal communication. In addition, attention is focused on the possible utilization of this procedure in dealing with certain craniopharyngiomas and other massive growths that completely usurp or distort the mid-supratentorial ventricle.

CASE REPORTS

Case 1. A 29-year-old white female, admitted Oct. 18, 1948, presented a 2-year history of personality changes, headache, amenorrhea, weight gain and progressive visual impairment. She also had symptoms of diabetes insipidus accompanied by urinary indifference. Examination revealed obesity, an obvious impairment of intellectual function and bilateral primary optic atrophy with visual acuity reduced to light perception only. Ventriculography demonstrated elevation and incomplete filling of the 3rd ventricle (Fig. 1). Bifrontal craniotomy was performed and a subtotal removal of an almost completely cystic adamantinoma was accomplished with improvement in the patient's visual acuity and fields and relief of headaches. Improvement continued for 2 months and then marked evidences of an obstructive hydrocephalus developed, necessitating her readmission. Ventricular estimation and drainage revealed a bilateral non-communicating hydrocephalus of the lateral ventricles. Bilateral ventriculocisternostomy was performed and the patient was discharged markedly improved. The postoperative

FIG. 1. Case 1. Distortion of 3rd ventricle produced by craniopharyngioma.

* Presented at the meeting of the Harvey Cushing Society, June 2, 1949, New Haven, Connecticut.
† Present address: Iowa State University Hospital, Iowa City, Iowa.

course was temporarily one of improvement but ultimately terminated fatally during the 4th month. Although the cause of death was unknown one would infer from historical data available that it was due to midbrain compression.

Comment. The ventriculographic studies revealed a suprasellar lesion which might well eventually lead to a bilateral occlusion of the foramina of Monro. The symptoms prompting readmission were primarily those of an obstructive hydrocephalus and with the disclosure of a bilateral non-communicating hydrocephalus of the lateral ventricles, a bilateral occlusion of the foramina of Monro appeared a reasonable explanation for the rapid deterioration in the patient's condition. Obviously, ventricular dilatation had not existed a long enough period of time to expect rupture of the septum pellucidum, so that a bilateral Torkildsen procedure appeared justified.

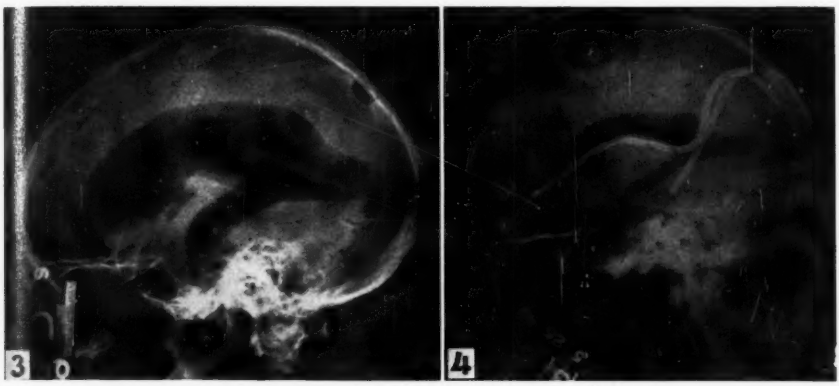
Case 2. A 20-year-old white female, admitted Dec. 3, 1948, complained of marked menstrual irregularities since onset of menstruation at age 14 and of rapidly failing vision. Headaches and occasional periods of nausea and vomiting had existed for 6 months. Positive findings were confined to the examination of the eyes. There was present a bilateral acute papilledema of 5 D. and reduction of visual acuity to gross finger movement at 4 feet. Roentgenograms of the skull demonstrated evidences of a long-standing hydrocephalus with a calcified area measuring 3×2×1.5 cm. in and superior to the sella turcica. Bifrontal craniotomy disclosed a firm, calcified, non-demarcated adamantinoma, densely adherent to the optic chiasm and tracts. A piecemeal removal of the tumor accomplished little in decompressing the chiasm. The postoperative course was stormy and because of persisting papilledema and stupor, a ventricular shunting procedure was deemed necessary. The finding of a severe bilateral non-communicating hydrocephalus on ventriculographic studies prompted a bilateral Torkildsen procedure (Fig. 2). Postoperatively, there was prompt subsidence of the papilledema, and relief of headache and somnolence. The visual acuity 4 months later was recorded at 2/300 O.D. and 2/1000 O.S., a distinct improvement over the preoperative findings on the right.

FIG. 2. Case 2. Bilateral ventriculocisternostomy tubes in place. The suprasellar calcification present was found at operation to represent less than ½ the actual tumor size, thus illustrating the degree of encroachment on the subthalamic region.

Comment. Ventriculographic studies, because of the obvious pathology present, were not considered necessary. Because of the fact that the primary problem was one of preserving vision and in view of the probable existence of a non-cystic lesion, a primary approach to the neoplasm was carried out but

unfortunately little was accomplished. As in Case 1, it was evident that both foramina of Monro were occluded and a bilateral ventriculocisternostomy was necessary.

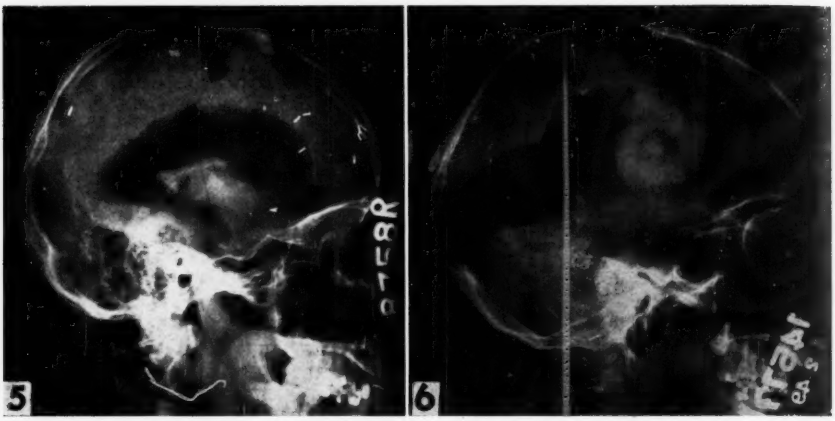
Case 3. A 39-year-old veteran, admitted Jan. 10, 1949, gave a 6-year history of increasing adiposity, thirst, sleepiness, headaches and visual disturbances. Later there developed diplopia, vertigo, continuous nausea and vomiting and staggering gait. Libido and sexual activity had never developed. Examination revealed a high grade of papilledema bilaterally, paresis of the left abducens nerve, bilateral partial loss of the lower visual field quadrants and diminution of visual acuity. Roentgenograms of the skull showed a large decalcified sella turcica and ventriculographic studies revealed a dilatation of both lateral ventricles with compression of the entire 3rd ventricle (Fig. 3). Because of the patient's precarious condition, the



FIGS. 3 and 4. Case 3. Severe dilatation of lateral ventricles and distortion of 3rd ventricle. Bilateral ventriculocisternostomy has resulted in a marked reduction of size of the lateral ventricles. The connecting Y-tube draining the two catheters is of non-radiopaque material and therefore not visualized.

evidences of severe increase in intracranial pressure and the long history, it was thought advisable to first decompress him. The tumor could then be removed at a later date when his condition permitted. It was expected that the tumor in expanding would obstruct both foramina of Monro and accordingly a bilateral ventriculocisternostomy was performed (Fig. 4). Thereafter the patient's condition improved rapidly and even before roentgenotherapy was instituted his visual fields assumed a normal range. Although the tumor itself has as yet not been surgically attacked the patient is now asymptomatic other than for symptoms of dyspituitarism and the visual acuity is now reported as 20/30.

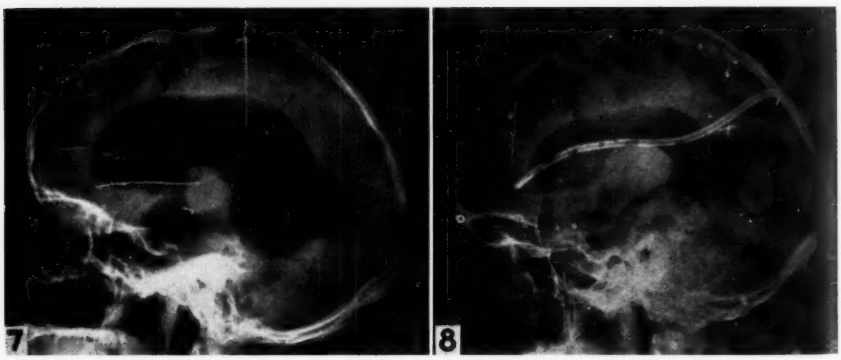
Comment. The ventricular studies demonstrated that the tumor was extremely large and that it had encroached upon the subthalamic structures and midbrain, obstructing completely the posterior portion of the 3rd ventricle. Only a small portion of the neoplasm could conceivably have been removed surgically and the obstruction might not have been relieved. In addition, his general condition was such as to constitute an extremely hazard-



FIGS. 5 and 6. A recurrent craniopharyngioma and an astrocytoma (Grade I) arising from the septum pellucidum and extending into the 3rd ventricle, producing in each case an obstruction of the foramina of Monro. The operative fatalities which resulted following primary surgery could, we believe, have probably been averted by a preliminary ventricular decompression by utilization of the bilateral Torkildsen procedure.

ous surgical risk. It is quite evident that the ventricular shunting procedure saved the life of the patient and produced a remarkable improvement in vision, both in the fields as well as the acuity.

In reviewing the histories, findings and the fatal outcome of other patients with extensive tumors involving the body of the 3rd ventricle and the interventricular foramen, we came to the conclusion that in certain cases a preliminary bilateral ventricular decompression would have relieved the



FIGS. 7 and 8. Posterior 3rd ventricle neoplasm. The intraventricular hypertension has resulted in a spontaneous rupture of the medial and posterior portion of the left lateral ventricle. Following a unilateral Torkildsen operation the ventricular dilatation has been partially reduced but the progressive increase in size of the tumor has completely obstructed the foramina of Monro. For comparative reduction in ventricular size in bilateral drainage versus unilateral, see Fig. 4.

acute symptoms and probably have improved the operative possibilities of a later removal of the tumor. Such could certainly have been the situation in a case of a recurrent craniopharyngioma (Fig. 5), and a large septum pellucidum astrocytoma extending from the inferior surface of the corpus callosum to the floor of the 3rd ventricle (Fig. 6). In an additional case of a posterior 3rd ventricle tumor with marked communicating hydrocephalus (Fig. 7), a unilateral Torkildsen procedure was performed and at a later date encephalographic studies (Fig. 8) revealed a decrease in the hydrocephalus but an increase in the size of the tumor with an almost complete obliteration of the foramina of Monro. A bilateral ventriculocisternostomy was thought to be imperative but subsequent roentgen therapy reduced the size of the tumor and relieved the foraminal obstruction, making this procedure unnecessary for the moment. However, in the event of recurrence, a second tube might aid in prolonging the life of the patient. This procedure, therefore, can be applied only to a few individual cases in which it is not expected that a direct attack upon the tumor would save the life of the patient or relieve the symptoms of ventricular obstruction.

DISCUSSION

If one reviews the literature on the surgical approach to the craniopharyngiomas, one is immediately impressed with the uniformly poor results that follow. In those tumors that are non-cystic one is almost never able to accomplish the removal of any sizable portion of the mass, and in the cystic lesions one's efforts are frequently defeated by the tendency for the cyst to refill in even as short a period as 1 month. Certainly, the ventriculocisternostomy will accomplish ventricular decompression more adequately and with less surgical risk than will a direct attack on the lesion in most instances. In Case 2 of this report, the ophthalmoscopic findings, which had apparently remained stationary following primary surgery directed toward the tumor, promptly began to regress following the shunting procedure. The visual disturbances of Case 3 also improved immediately following this operation. It is contemplated in our future dealings with the craniopharyngiomas presenting an obstructive hydrocephalus to consider a preliminary Torkildsen procedure, either unilateral or bilateral, depending upon the ventriculographic findings.

The idea of the bilateral ventriculocisternostomy is not an original one. In his monograph, Torkildsen suggested that a bilateral procedure might be necessary in some instances where both foramina of Monro were occluded. He pointed out that this possibility must be rare because of the tendency for the septum pellucidum to rupture and to establish a new communication between the two cavities. However, his experiences in one case (Case XIX) demonstrates that a unilateral increase in intracranial pressure may have fatal consequences before rupture of the septum pellucidum occurs. It is felt that a similar situation existed in Case 1 of this report in that with both ventricles decompressed a catastrophe was averted. In a recent personal

communication, Torkildsen[3] indicated that he had carried out bilateral insertion of tubes. His experiences being limited, he did not attempt to discuss this deviation from his original method.

CONCLUSION

It is suggested that in certain space-occupying growths within or adjacent to the 3rd ventricle with occlusion of both foramina of Monro, a bilateral ventriculocisternostomy might be the procedure of choice.

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EXPERIENCES WITH UNILATERAL PREFRONTAL LOBOTOMIES FOR PAIN*

STUART N. ROWE, M.D., AND JOHN B. MOYAR, M.D.

Neurosurgical Services of the Veterans Facility, Aspinwall, Pennsylvania, and Western Pennsylvania Hospital, Presbyterian Hospital and St. Francis Hospital, Pittsburgh, Pennsylvania

(Received for publication June 14, 1949)

In 1945 Freeman and Watts[1] suggested the use of frontal lobotomy for the relief of intractable pain and the procedure has gradually achieved rather general recognition and use.[2,3,5] Koskoff *et al.*[4] in December 1947 reported on a limited experience with unilateral operation for the same purpose and recommended that it be tried, initially at least, in patients with advanced malignancy and poor physical condition. In March 1948 Scarff[6] described his immediate results in 4 cases of unilateral lobotomy for pain. In all of these relief was obtained for the short period of the follow-up studies. The operation was effective whether carried out on the side of the pain or on the opposite side, and altered the mental status very little. Subsequently, Scarff[7] reported upon a total of 10 cases with follow-ups from 6 weeks to 6 months after operation. In this group the results were good in 7. It was his impression that the poor results were obtained when the operation was carried out on the non-dominant hemisphere for contralateral pain or in those instances in which pain was due to involvement of the cranial nerves or the dura mater. On the basis of these encouraging reports unilateral leucotomy was carried out in 16 cases of intractable pain. This series forms the basis of the present paper.

SELECTION OF CASES

In selecting cases for this study, an effort was made to obtain patients in a wide age scale as well as patients with malignant and non-malignant lesions. Four cases are of interest in that they deal with pain involving cranial nerves. In 1 it was due to bilateral trigeminal neuralgia, and in the others it resulted from carcinoma involving the trigeminal and glossopharyngeal nerves on the left. Two paraplegic patients whose lesions were of the cauda equina suffered from severe radicular pain. One of these incurred his injury from multiple shrapnel wounds, and numerous metallic fragments, as well as extensive trauma to the upper lumbar vertebrae, could be demonstrated on x-ray (Fig. 1). Twelve patients in the series had advanced malignancy with metastases, and 1 patient suffered from pain secondary to severe spasticity of a congenital diplegia.

* Presented before The Academy of Neurological Surgery, October 26, 1949, Portland, Oregon.
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TECHNIQUE

Despite the poor general condition of several of these patients (two-thirds of the group suffered from advanced malignancy), operation was accompanied by very little shock with the single exception mentioned below, and supportive measures were not required. In 5 instances the procedure was carried out under local anesthesia.

The first patient in the group was operated upon in the sitting position with the thought of minimizing bleeding and cerebral edema. His condition was poor and he expired during operation, apparently as a result of cerebral anoxemia. All subsequent operations were done with the patient supine with

FIG. 1. Case 7. Residual metallic fragments and bone changes associated with cauda equina injury and intractable radicular pain.

the head slightly elevated. The superior approach was used with a scalp incision to expose the coronal suture 3 cm. lateral to the midline. A $1\frac{3}{4}$ inch trephine opening was made and after the reflection of a dural flap posteriorly the cortical vessels were coagulated. A cannula was passed to the lesser wing of the sphenoid bone to establish a track for the frontal lobe incision (Fig. 2). The plane of this incision thus extended from the coronal suture (usually about 13 cm. posterior to the glabella) to the posterior margin of the lesser wing of the sphenoid bone, in most cases just entering the tip of the lateral ventricle (Fig. 3). The cut was made under direct vision using a small suction tip and lighted retractor. Inferiorly and medially the incision reached the gray matter and it was carried laterally 4 to 5 cm. (Fig. 4).

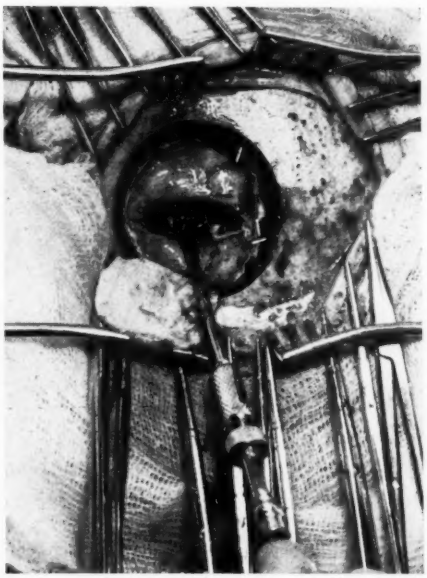
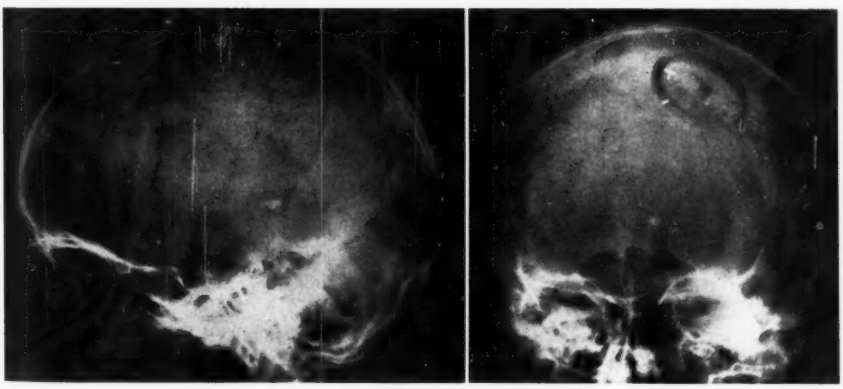


FIG. 2. Case 9. Locating the posterior margin of the lesser wing of the sphenoid bone. The cannula enters the brain in the plane of the coronal suture and 3 cm. lateral to the midline.

In the first 9 patients no special effort was made to cut the superior lateral quadrant. This was in part due to our impression that the medial portion of the incision was the most vital and was in part the mechanical result of making an incision from a superior approach with a straight instrument. In an effort to improve our results the frontal lobe incision was enlarged to include the superior lateral quadrant by using a curved probe and carrying the section to the gray matter. In the last 7 cases the lobotomy was carried out on the left side in this manner.

RESULTS

In the first 9 cases (Table 1) the results of unilateral lobotomy were somewhat disappointing. Only 1 patient (Case 3) obtained complete relief of pain, which persisted until her death 1½ months later. This result was particularly impressive in view of a spinal metastasis with accompanying kyphosis and severe root pain. The relief in most of the remaining cases was complete for the first week, following which the original evidences and complaints



FIGS. 3 and 4. Case 9. *Left*, A lateral roentgenogram of a steel wire inserted into the frontal lobe incision demonstrates the plane of the lobotomy. *Right*, An AP view taken to demonstrate the lateral extent of the lobotomy. It was felt that the wire did not reach the lateral margin of the incision.

TABLE 1

Case	Age	Diagnosis	Type of Pain	Operation	Result	Subsequent Operation	Result	F.U.
1. L. J. K.	40	Bronchogenic carcinoma	Chest & brachial plexus, rt.	R. lobotomy	Died at op.			
2. E. C.	55	Carcinoma mouth	Mouth, throat, ear, lt., & slight on rt.	R. lobotomy	Relief 1 mo.	Gasserian ganglion injection	Relief. Went home. Recurrence of pain	3 mos. 4 mos.
3. V. L.	39	Metastatic carcinoma breast	Arm, chest & spine. Bilateral radicular	R. lobotomy	Relief. Marked mental dulling		Died 1½ mos. Wound infection	
4. R. O.	29	Congenital diplegia. Spasticity	Generalized muscle & joint	R. lobotomy	Relief 10 days	L. lobotomy	Relief	3 wks.
5. F. C.	20	Bilateral trigeminal neuralgia	Bilateral paroxysmal, face	R. lobotomy	Slight relief	Trigeminal rhizotomies	Relief	4 mos.
6. M. M.	42	Uterine carcinoma. Pelvic metastases	Pelvic & cauda equina	R. lobotomy	Relief 2 wks.	L. lobotomy	Relief. Died of carcinoma	1½ mos.
7. W. R. J.	26	Traumatic myelitis—cauda equina	Paroxysmal radicular, legs	L. lobotomy	Relief 1 wk.	Unilateral cordotomy	Relief	3 mos.
8. H. H.	25	Traumatic myelitis—cauda equina	Radicular, legs. Phantom limb, rt.	L. lobotomy	Moderate relief	Bilateral cordotomy	2 convulsions. Wound infection	3 mos.
9. F. O. W.	56	Carcinoma pancreas	Severe epigastric radiating into back	L. lobotomy	Incomplete relief	R. lobotomy	Complete relief after 2nd operation	2 mos.
10. E. M.	45	Malignant melanoma	Lt. shoulder & arm	L. lobotomy	Relief		Discharged home	4 mos.
11. R. S.	58	Bronchogenic carcinoma	Chest & lt. arm	L. lobotomy	Relief		Died pneumonia	3 wks.
12. L. C.	55	Carcinoma left antrum	Facial & orbital	L. lobotomy	Relief			2 mos.
13. E. F.	54	Metastatic carcinoma cervical spine	Shoulder & arm	L. lobotomy	Relief		Discharged	3 mos.
14. E. G.	21	Malignancy kidney	Chest & abdominal	L. lobotomy	Relief		Died at home	1 mo.
15. J. H.	61	Carcinoma parotid	Face	L. lobotomy	Relief		Died in hospital—cardiac?	1 wk.
16. C. E.	51	Carcinoma pancreas	Abdominal	L. lobotomy	Relief			1 mo.

of suffering recurred and narcotics had to be resumed. In 2 cases more prolonged but only partial relief was obtained. Five patients required additional surgical procedures to control their pain, including lobotomy on the remaining side in 2 instances. Following these operations relief of pain and freedom from the use of narcotics were obtained in all instances.

The last 7 patients, all treated by nearly complete left frontal lobotomy, obtained very satisfactory relief of pain. It is interesting to note that 2 of these suffered from involvement of branches of the trigeminal nerve and the other 5 from invasion of the brachial plexus. Previous experience suggested that pain from invasion of cranial or peripheral nerves was not easily amenable to lobotomy.

Following unilateral lobotomy fairly marked mental changes were usually observed for about 1 week to 10 days. These included moderate mental dulling, loss of inhibitions, flattening of affect, impairment of recent memory and dissociation of anxiety from pain. Even during this period, however, incontinence of urine or feces occurred in only 1 patient in this group. In several of the patients the improvement in morale was striking and it was accompanied by an improved appetite and gain in weight. Withdrawal symptoms were not observed even though most patients had been accustomed to large doses of narcotics. In some patients the resumption of a normal mental status was accompanied by the reappearance of pain.

Two complications occurred in this series. Two patients developed superficial infections as a result of their scratching the wounds. These healed with the aid of antibiotics and local therapy. One patient had two generalized convulsions within the first 4 days after operation. He was placed on dilantin therapy and has had no further seizures. EEG postoperatively revealed random spikes over the operated frontal lobe.

COMMENT

The advantages of unilateral leucotomy over the bilateral operation are readily apparent. The operative shock is negligible, permitting its use even in debilitated patients, the postoperative convalescence is short, and the mental changes are mild and temporary. Four patients examined with a battery of mental tests showed no significant change between the pre- and postoperative studies.

We are still somewhat in doubt as to the efficacy of the unilateral operation. The results in our first 9 patients were disappointing since only 1 obtained complete and lasting pain relief; however, in the last 7 patients a more complete incision was made in the dominant frontal lobe and gratifying control of pain followed.

Certainly it is not difficult to add the operation on the second side to a unilateral lobotomy if recurrence of pain requires it. With this thought in mind and the experience of very satisfactory results in one-half of the patients treated with operation on one side, we plan to continue our practice of using this procedure for intractable pain.

SUMMARY

1. In a series of 16 cases of intractable pain treated by unilateral lobotomy, lasting relief was obtained in 50 per cent of the series.

2. In some instances at least, unilateral lobotomy gives effective relief when the pain is diffuse, when it is due to direct nerve involvement, or when it occurs in the upper extremities or head.

3. Our experience has suggested that complete unilateral leucotomy is more effective than a medial incision that does not include the superior lateral quadrant.

4. The most impressive advantages of the unilateral operation are the minimal surgical risk and the absence of marked postoperative mental change.

5. Pain was subsequently controlled satisfactorily by making the operation bilateral in 3 cases. Other pain-relieving operations were successfully used in several additional cases.

6. On the basis of this small series of cases, we feel that unilateral lobotomy has a definite place in the treatment of intractable pain and deserves further trial. Subsequent operation on the second side may always be added if necessary.

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CEREBRAL ANGIOGRAPHY IN "BRAIN TUMOR SUSPECTS"

GEORGE G. CULBRETH, M.D., A. EARL WALKER, M.D., AND ROBERT W. CURRY, M.D.

Division of Neurological Surgery, and Department of Radiology, The Johns Hopkins University School of Medicine, Baltimore, Maryland

(Received for publication July 7, 1949)

THIS is a report of the angiographic findings in a consecutive series of "brain tumor suspects" seen at the Johns Hopkins Hospital during a period of approximately one year. While the concept of "brain tumor suspect" is rather vague, we have used the term to include all cases in which diagnostic procedures such as pneumoencephalography, ventriculography or arteriography were carried out to eliminate the possibility of an intracranial neoplasm. We realize that our index of suspicion is rather high and that at times such procedures were done when we thought the possibility of neoplasm remote. Some patients, had they been seen on the average neurological service, would probably have been classified arbitrarily as having cerebrovascular disease and discharged without the benefit of these techniques. For example, we have made such special examinations in all cases of convulsive seizures occurring for the first time in adult life.

TECHNIQUE OF ARTERIOGRAPHY

Percutaneous puncture of the carotid artery was practiced after infiltration of the skin of the neck with procaine. If the vessel could not be cannulated within a reasonable time, an incision was made and the vessel injected by direct vision. In this series of cases, the carotid artery was successfully punctured percutaneously in 92 of the 96 patients.

The roentgenograms were made in series of 3 films, taken at 1 sec. intervals after the injection of 15 cc. of 35 per cent diodrast. Stereoscopic views were obtained by shifting the x-ray tube and repeating the procedure. As a routine, stereoscopic frontal and lateral projections were made, necessitating 4 injections of the contrast media. The 3 serial films usually demonstrated arterial, capillary and venous phases of the angiogram. There appeared to be great variation in the cerebral circulatory time. Occasionally the arterial phase of the cerebral circulation coincided with the capillary phase of the circulation within the neoplasm; at times the contrast medium remained in the tumor after it had disappeared from the cerebral vessels. These variations in cerebral circulatory time made it necessary to have serial films for good visualization of the intracranial vessels.

During the injection of the diodrast, the patient usually complained of a pain in the neck or head and occasionally jerked or jumped. With proper preparation, however, few complications occurred. Transient hemiparesis and/or aphasia has been seen in 4 patients of this series following angiog-

raphy. In only 1 case of suspected cerebral thrombosis could the procedure have been contributory to the patient's demise.

RESULTS OF ANGIOGRAPHY

Some 96 "brain tumor suspects" have been subjected to angiography. Of the 93 successful angiograms, we have been able to verify the diagnosis by subsequent operation or autopsy in 54 cases. In 8 cases the angiographic findings were indicative of a lesion (in 7 instances neoplasm, in 1 porencephaly) but for medical or personal reasons it was not verified; in 31 cases the arteriogram was interpreted as normal, and in 24 of these cases in which air studies were made, pneumoencephalograms did not suggest a space-occupying lesion. In 1 patient having a normal angiogram, a thalamic tumor was demonstrated by an air study.

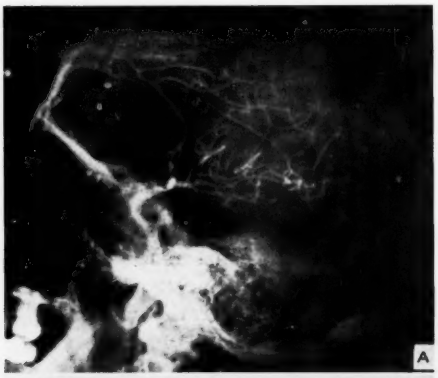

FIG. 1. A. (*O. A.*) Lateral angiogram (arterial phase) showing a large meningioma of the left olfactory groove displacing the anterior cerebral artery superiorly and posteriorly. A hyperostosis of the tuberculum sellae is apparent. B. (*A. H.*) Lateral angiogram (arterial phase) demonstrating a central astrocytoma displacing superiorly the anterior cerebral and inferiorly the middle cerebral arteries. Few vessels can be seen within the tumor.

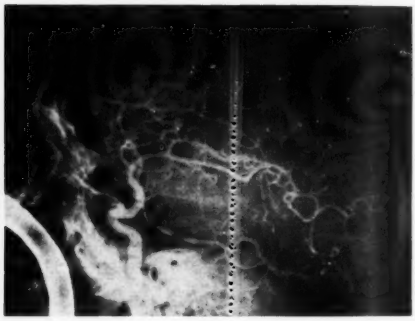

FIG. 2. (*W. D.*) Lateral angiogram (arterial phase) to show the superior displacement of the middle cerebral arteries by a temporal lobe astrocytoma. The supraclinoid curve of the carotid siphon is opened.

LOCALIZATION OF TUMOR BY ANGIOGRAPHY

Angiograms may indicate the presence of a space-occupying lesion by distortion of normal vascular structures or by showing the intrinsic vascular pattern of the tumor.[7,18,19,22,23,27,28,29,30,32,33,41,45,48,50]

The site of an intracranial neoplasm may be determined by the displacement of the normal vascular supply of the involved area. In the frontal region, lateral displacement, elevation or depression of the anterior cerebral artery are the usual signs of a space-occupying lesion (Fig. 1A).[14] In the central region, lateral displacement of the anterior cerebral artery and depression of the middle cerebral artery indicate a neoplasm (Fig. 1B). In the temporal lobe, distortion of the carotid siphon, elevation of the middle cerebral artery and lateral displacement of the anterior cerebral artery are common evidence of a tumor (Fig. 2). In the parietal lobe, depression of the middle cerebral artery is the most common finding. Tumors of the occipital lobe often do not distort carotid angiograms unless the posterior cerebral artery is filled fortuitously. Tumors at the base of the brain produce abnormalities in the shape of the carotid siphon, displacement of the middle or anterior cerebral arteries and/or compression of one of these vessels[36] (Table 1). Tumors of the posterior fossa, as originally noted by Moniz and Alves[37] may cause elevation of the posterior cerebral artery (Fig. 3A).

TABLE 1

Angiographic Findings in Sellar and Parasellar Tumors

	Craniopharyngiomas	Pituitary Adenomas	Meningiomas
Compression of carotid siphon	0	1	0
Displaced 1st curve carotid siphon	4	4	1
Displaced supra-clinoid portion carotid siphon	2	1	1
Displaced middle cerebral	1	1	1
Displaced anterior cerebral	2	0	0
Compression of artery	4	0	1
Total number of cases	5	4	1

In the entire group of 42 verified brain tumors, in only 3 instances was the lesion not localized by angiography. Two of these cases were neoplasms of the medio-inferior aspect of the temporal lobe. These patients had initial ventriculograms which were interpreted as normal. In both, on subsequent air studies, the ventricles were distorted by the tumor. In the case of the third normal angiogram two small meningiomas, localized by the type of focal epilepsy, were found over the motor cortex.

In 2 cases a space-occupying lesion was assumed from angiograms, but at operation found to be edema associated with vascular thrombosis.

ANGIOGRAPHIC CHARACTERISTICS OF TUMOR TYPES

The angioarchitectural characteristics of an intracranial tumor have been considered sufficiently specific to warrant a pathological diagnosis[16,20,25,26,35,39,47] (Table 2). Four types of tumors are said to have specific vascular patterns.

I. *Vascular Malformations and Tumors*

It is to be expected that angiomatous malformations and tumors would be spectacularly demonstrated by angiography. The degree of visualization of such anomalies depends upon the type and extent of the lesion. The angiomatous conditions are divided into true tumors (hemangioblastomas) and vascular malformations. To the best of our knowledge no report has been made of the angiographic appearance of the former, which commonly occur in the posterior fossa. Green and Arana[17] show the carotid angiogram of a

TABLE 2

Frequency of Angiographic Characteristics of Cerebral Tumors

	Astrocytomas	Glioblastomas	Meningiomas
Displaced cerebral vessels	4	13	9
Intrinsic vascularity			
Increased	1	11	7
Decreased	3	1	2
Loops and sinuses	0	8	3
Total cases	4	14	11

patient who they considered had a cerebral hemangioblastoma but the lesion was not verified by histological inspection.

The usual classification of malformations may be abbreviated for practical purposes as follows:

1. Cavernous angioma
2. Angioma racemosum
   a. Telangiectasis
   b. Venous angioma
   c. Arteriovenous angioma

Cavernous Angioma. These tumors, differentiated from the malformations by the fact that nervous parenchyma is not present between the walls of the vascular sinuses, are quite rare and so far as we have been able to ascertain, have not been demonstrated by angiography.

Telangiectasis. The common site for telangiectasis is the lower brain stem,[6] which can be visualized but poorly by angiography. Telangiectatic lesions of the cerebral hemispheres are usually associated with some other evidence of phakomatosis. Owing to the small size of the vessels involved in this type of malformation and the poor resolution by the x-rays, it is difficult to demonstrate the lesion by carotid arteriography.* Both Engeset[13] and we have attempted the demonstration of the vascular telangiectasis

* Although the literature has many references to visualization of individual capillaries by angiography it is impossible by the usual x-ray techniques to resolve an object smaller than 0.1 mm. in diameter. Since capillaries are generally considered to have a diameter less than 50 μ, it is obvious that individual capillaries cannot be seen in angiograms. The "capillaries" referred to probably represent arterioles 0.3–0.5 mm. in diameter.

associated with Sturge Weber syndrome without success. Green and Arana[17] state that in one case of this syndrome "peripheral capillary loops" and "abnormal blood vessels of small calibre" were incorporated in the calcified area of the cerebrum.

Venous Angiomas. No angiograms of this rare type of cerebral varix have been reported to our knowledge.

Arteriovenous Angiomas. This is the most common type of angiomatous malformation encountered and the most favorable for angiographic demonstration.[12,16,17,24,34,46] Small arteriovenous angiomas, consisting of a few tortuous or coiled vessels, such as illustrated by Govons and Grant in their Fig. 8[16] and such as we have seen, are asymptomatic until a rupture occurs. The larger angiomatous malformations, commonly the basis of such neurological phenomena as epilepsy, hemiparesis or hemianopsia, may involve a considerable part of one lobe of the brain.

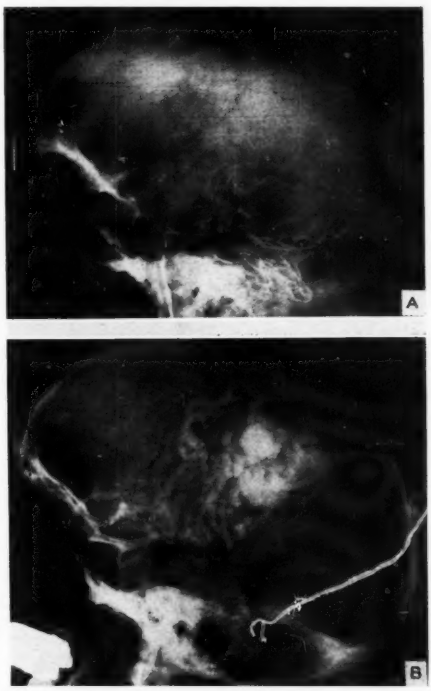

FIG. 3. A. (*L. H.*) Arterial phase of a lateral vertebral angiogram showing a slight superior displacement of the left posterior cerebral artery due to a meningioma attached to the tentorium. The right posterior cerebral artery lies in its normal position. B. (*P. S.*) Lateral angiogram (late arterial phase) demonstrating an angioma of the right central region.

As expected from the pathological anatomy of these angiomas,[6] angiomas of the arteriovenous malformations of the brain demonstrate enormous channels and sinuses fed by abnormally large, tortuous arteries and drained by hugely dilated veins. The arteries supplying the area are both more numerous and of larger calibre than normal, a fact which Tönnis[46] and others have noted (Fig. 3B). The circulation of these angiomatous malformations appears to be unusually brisk because of the fistulous communications between arterial and venous channels, and accordingly the lesion is best visualized during the arterial phase of the injection. Yet there appear to be sinuses, perhaps diverticulae, in the malformation from which the blood slowly escapes, for small circular collections of diodrast are often seen within the lesion during the venous phase of the cerebral circulation. The calibre of the venous channels leading

from the malformation is much greater than that of the entering vessels. Angiography usually visualizes well the angiomatous lesion but rarely the normal vessels irrigating the cerebral hemisphere. From the surgical standpoint this finding is important as it suggests that the vascular supply of the cerebral structures is derived from the opposite carotid artery through collateral communicating channels. Thus if ligation of the carotid artery on the side of the lesion becomes necessary, there is presumptive evidence that the cerebral circulation will remain adequate.

II. *Glioblastomas*

That glioblastomas have a specific angioarchitecture has been recognized and described by many neuropathologists.[2,4,15,42] Although Bailey and Cushing[2] mention the vascular changes in these tumors, a more complete angioarchitectural picture has been given by Deery[9,10] and Hardman,[20] who

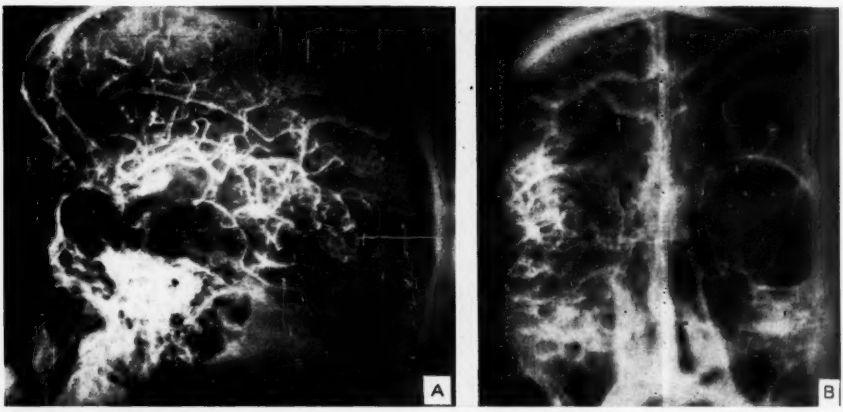

FIG. 4. (*J. M.*) A. Lateral angiogram (late arterial phase) demonstrating the vascular pattern of numerous loops and sinuses in a right parietal glioblastoma. B. The frontal angiogram (capillary phase) suggests that the tumor does not involve the basal ganglia.

studied sections taken through the entire tumor. Both of these papers emphasize that the vascular pattern varies from the periphery to the centre of the tumor. In general the capillaries on the periphery of the tumor are dilated and tortuous. In the margin of the actively growing tumor these dilated capillaries have localized dilatations or diverticuli which may serve as arteriovenous fistulae. These aneurysmal sinuses frequently have adventitial thickening and internal proliferation which may be associated with thrombosis. Toward the central portion of the tumor the vessel walls are hyalinized, the lumen closed by thrombi and the tumor tissue necrotic, cystic and undergoing fibrosis. These pathological changes in the different parts of the tumor may be merely temporal stages in the neoplastic development. It should be stated that, although the above description applies to perhaps

two-thirds of the glioblastomas, there are many tumors that do not exhibit such a typical vascular pattern. We would presume that some of the difficulty in classifying a tumor as "angionecrotic,"[3] "angiothrombotic" or "angioproliferative"[8] arises from the fact that biopsy specimens, often small and from only one part of the periphery of a tumor, do not give the complete angioarchitectural picture of the neoplasm.

With this knowledge of the pathological anatomy of these tumors it would be expected that the angiographic picture of the majority of glioblastomas would be characterized by increased vascularity in the margin of the tumor with sinuses, irregular dilatations and vascular loops within the neoplasm (Fig. 4). This is the typical finding in glioblastomas,[13,17,26,38] and according to Busch and Christensen[3] it is suggestive of the angionecrotic type of tumor. This angiographic picture is present in from 53 per cent[31] to 78.5 per cent[17] of glioblastomas. In some angiograms, the typical angioarchitecture, peripheral vascularity to central necrosis (Fig. 5A), is well illustrated. In other cases the increased vascularity may be diffuse and resemble that seen in meningiomas. At times, the tumor may appear to be practically avascular except for one small area of fine angiomatous proliferation. Such gliomas, Engeset[13] emphasizes, should be classified as glioblastomas or as astrocytomas undergoing malignant change.

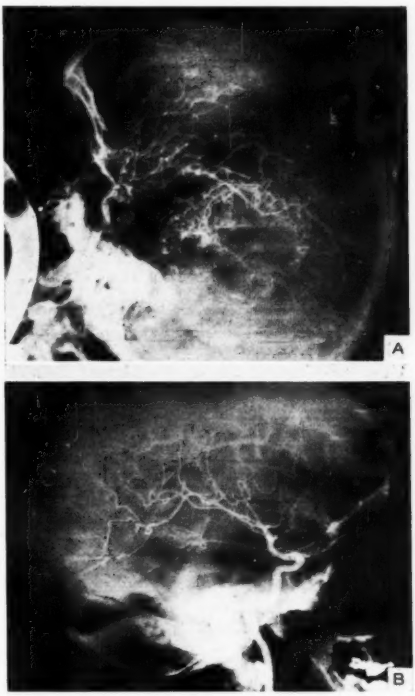

FIG. 5. A. (*V. R.*) A lateral angiogram (late arterial phase) to show the vascular pattern of a large parietal lobe glioblastoma. B. (*M. M.*) Lateral angiogram (arterial phase) to demonstrate the vascular distortion produced by an astrocytoma of the right temporal lobe. Except for one surface artery the tumor appears avascular.

III. *Astrocytomas*

It is generally agreed that astrocytomas are relatively avascular tumors. Elsberg and Hare[11] found that in less than half of the astrocytomas examined there were grossly visible blood vessels in the deeper part of the growth and rarely discernible vessels in the periphery. Using a benzidine stain Sahs and

Alexander[44] found that the astrocytomas had the least density of vessels, the smallest calibre of vessel and the most regular of sinusoids of all the glial tumors. Bailey[1] states that the small vessels of astrocytomas are frequently calcified.

It is not surprising then that Engeset[13] considers the angiographic char-

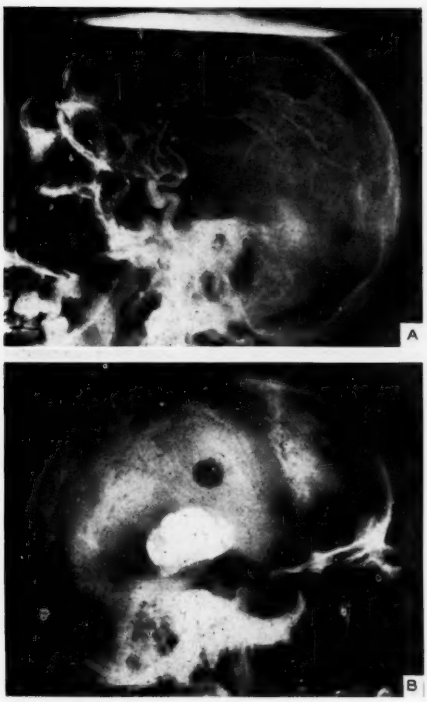

FIG. 6

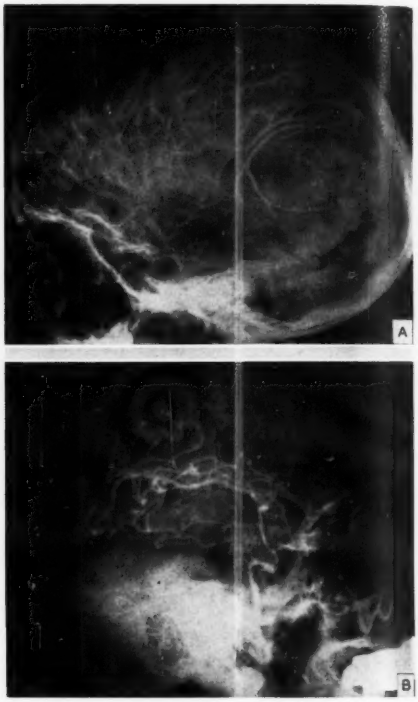

FIG. 7

FIG. 6. (*V. C.*) A. Lateral angiograms (arterial phase) showing the displacement of the middle cerebral vessels by a cystic astrocytoma of the temporal lobe. B. Visualization of the cyst by thorotrast.

FIG. 7. A. (*W. C.*) Lateral angiogram (late arterial phase) of a large parietal meningioma which is outlined by the vessels about the tumor. B. (*R. R.*) A lateral angiogram (arterial phase) to show the vascular pattern of a medial sphenoidal ridge meningioma and the distortion of the carotid siphon. The carotid artery is seen to be narrowed as it passes through the tumor.

acteristics of astrocytomas to be displacement of normal vessels by an avascular area or an area having few, fine, uniform straight vessels or, if the tumor is associated with a cyst, slender vessels curving about its margin. This is in agreement with the opinion of most previous investigators[13,21,26] and with our limited experience (Fig. 5B). But Moniz[34] considers that these tumors have a dilated vascular network. We agree with Engeset's comment

that Moniz's observation is probably the result of the inclusion of cases that should have been classified as glioblastomas.

The temporal lobe astrocytomas, as noted by Conrad[5] and Quarti,[43] produce a typical angiographic picture characterized by curved elevated middle cerebral arteries overlying a relatively avascular mass (Fig. 6). Complete avascularity suggests, in our experience, a cystic tumor.

IV. *Meningiomas*

The intrinsic vascular pattern of meningiomas varies so greatly that no typical angioarchitecture can be ascribed to it. The specificity of the angiographic picture is more dependent upon the morphological structure of the lesion than upon its intrinsic vessels. Because meningiomas are circumscribed, encapsulated tumors, clearly demarcated from the brain, they produce regular deformities in the normal cerebral angioarchitectonics, as originally described by Moniz[34] (Fig. 7A). Moreover, since the blood supply of the tumor frequently is derived from cortical vessels coursing over its capsule and arborizing within it, a basket angioarchitectural appearance with a centripetal spray or network is characteristic of the angiogram (Fig. 7B). The intrinsic vessels, usually of precapillary size, may be so closely packed that the tumor has the appearance of a solid or slightly mottled mass, the so-called "opacification" (Fig. 8A).

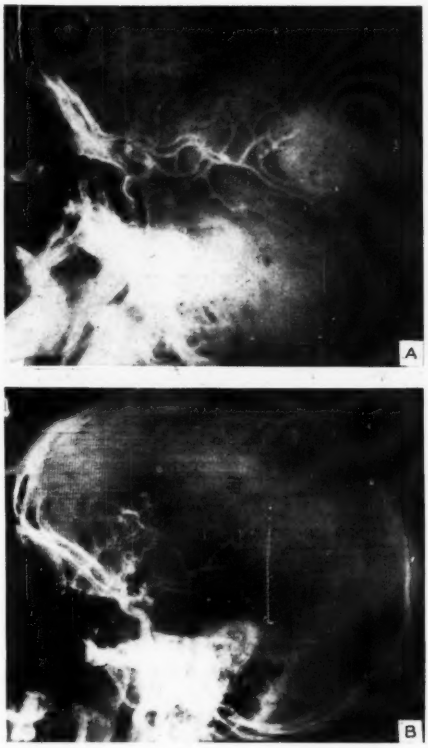

FIG. 8. A. (*F. Y.*) A lateral angiogram (arterial phase) to show the vascular pattern of a meningioma. Although only the arterial pattern of the brain is demonstrated, the capillary pattern of the tumor is seen as a uniform opacity. B. (*R. S.*) A lateral angiogram of sphenoidal ridge meningioma which resembles a glioblastoma.

Because the tumor invades the meninges and bone, its blood supply may be largely from the external carotid artery and the usual carotid angiogram may demonstrate only the displacement of major cerebral vessels. In some cases the vascular supply is from both internal and external carotid arteries.[26]

For these reasons, it is necessary to inject both carotid arteries with contrast media when a meningioma is suspected.

Although the usual angiogram of a meningioma presents a circumscribed area of increased vascularity, the outline of the tumor may be indistinct, as List and Hodges[26] have noted, and the angiogram resemble that of a vascular glioblastoma. In 3 meningiomas of our series the angioarchitectural pattern simulated that of a glioblastoma, but in only 1 of these cases was a differentiation impossible preoperatively (Fig. 8B).

V. *Other Types of Tumor*

Moniz,[34] Hemmingson[21] and Engeset[13] describe the angioarchitecture of the oligodendrogliomas as similar to that of the astrocytomas. Metastatic carcinoma has much the same angiographic appearance as the glioblastoma.[17,26] Lorenz[31] describes a case of meningeal sarcoma in which there was a rich double circulation with arteriovenous anastomoses.

VALUE OF ANGIOGRAPHY

It is apparent that angiography is of value both in localizing the site of a tumor and in indicating its probable pathological structure.

There is evidence to show that the technique is not infallible. Our experience with angiography in tumors of the posterior fossa has been so meagre that we cannot make a statement of its value. But in supratentorial tumors carotid angiography may fail to show tumors of the basal ganglia and midline structures, the inferior temporal region and the occipital region. For the demonstration of such tumors ventriculography is preferable to angiography.[49] In some 13 cases in this series both angiograms and ventriculograms were made either simultaneously or seriatim. In 11 instances the interpretations of the two procedures coincided; in 1 case, a thalamic tumor, the ventriculogram demonstrated the tumor but the arteriogram did not; and in 1 case the arteriogram was positive and the air study negative. In the majority of cases, air studies were not done because the arteriograms were adequate for localization of the tumor. Arteriography seemed to cause less discomfort to the patient than pneumoencephalography or ventriculography and had the distinct advantage that it did not aggravate the patient's symptoms necessitating an immediate craniotomy.

In planning a craniotomy angiography helps the surgeon by indicating the position of major arteries irrigating the neoplasm.[40] This is of particular value in vascular meningiomas and in some gliomas. This information is important in tumors about the sella turcica, where the internal carotid arteries are frequently involved in the tumor. With the knowledge that the internal carotid artery runs through a tumor, the surgeon is spared the humiliation of having a casualty from exsanguination and the patient, a fatal outcome.

SUMMARY

A series of 96 angiograms of "brain tumor suspects" is analyzed. In 3 cases the roentgenograms were technically unsatisfactory. Air studies or the

course of the disease, verified the angiographic diagnosis in all but 5 cases.

In the group of 42 verified brain tumors, with 3 exceptions, the angiograms were interpreted as correctly localizing the lesion. Tumors of the medial part of the temporal lobe and the midline structures failed to visualize by angiography.

Arteriovenous angiomas are characterized by a mass of tortuous channels and sinuses fed by large corkscrew arteries and drained by greatly dilated veins.

Approximately two-thirds of the glioblastomas have a typical angiographic appearance characterized by increased vascularity at the margin of the tumor with sinuses, irregular dilations and vascular loops within the tumor.

Astrocytomas have as angiographic characteristics the displacement of normal vessels by an avascular area or an area having few fine, uniform straight or slightly curved vessels.

Meningiomas do not have a typical intrinsic angioarchitecture, but the vascular abnormalities produced by their circumscribed nature give them specific characteristics.

Angiograms aid in the planning of surgical removal of the tumors and give information which may prevent surgical disasters.

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CEREBRAL ANGIOGRAPHY RECORDED CINEFLUOROGRAPHICALLY

HARVEY GASS, M.D.,* SYDNEY WEINBERG, ALBERT CRAIG, M.D., JOHN J. THOMPSON, M.D., AND FRANK DREISINGER, R.T.

Department of Surgery, Neurosurgical Division, Department of Radiology, and Department of Medicine, Strong Memorial Hospital and the University of Rochester School of Medicine and Dentistry, Rochester, New York

(Received for publication August 2, 1949)

THE purpose of this paper is to report progress in the development of a successful cinefluorographic method of cerebral arteriography.

Stationary records of the shadows of brain vessels as obtained in the conventional method of single or even repeated roentgen-ray exposures allow only glimpses of the vascular flow of a contrast medium. Although in many instances such glimpses reveal sufficient information for diagnosis, frequently, because of faulty timing, these glimpses are not enough. Efforts to overcome this deficiency have been largely in the direction of increasing the number of exposures during the injection, repeating the injection of the dye and secondary injections for stereoscopic views. Most of these efforts have further complicated the technique. The ultimate aim has been to obtain a continuous record of the flow of radiopaque dye through the vessels of the brain. The potential yield of information from such a record might go far beyond the identification of pathological vascular configurations.

The need for the development of such a technique has been recognized before but to our knowledge only one attempt at producing one for practical use has thus far been reported. This was by Holm[1] in 1944. He also used the indirect method of photographing the images on a fluorescent screen and took 8 or 16 frames per second. Although he was able to obtain some records he admitted that the quality of the films was not adequate to permit the clinician to rely on this technique alone and recommended its use as a complement to the conventional method. With the development at Strong Memorial Hospital of a practical cinefluorographic apparatus it has now become possible to make continuous cerebral arteriographic records that are satisfactory for clinical use. Earlier publications have described the technical construction of this apparatus in detail.[2]

We have employed this method thus far in obtaining 20 satisfactory angiograms. By preference we have used the percutaneous method exclusively although the direct surgical exposure technique is equally suitable.

The patient is first appropriately fixed in a recumbent position so that his head is immobilized adjacent to the screen. We use a specially built platform upon which the patient lies which has a prolongation at one end to which his head is secured with restraining straps (Fig. 1). If necessary a pillow is placed under his shoulders

* University Hospital, Ann Arbor, Michigan.

for slight hyperextension of the neck. The hands and knees are restrained. One person only is needed to make the puncture and the injection. The cinefluorographic apparatus can be operated by one technician.

In making the puncture we use an ordinary #18 lumbar puncture needle. The skin and subjacent tissues are first infiltrated with 1 per cent novocain down to the wall of the artery. We have found it extremely satisfactory then to give the patient an intravenous dose of sodium amytal varying between 0.3 and 0.6 gm. This is generally sufficient to render him amnesic or asleep for at least 10 to 15 min. so that he remembers nothing of the procedure when he awakens. As soon as the patient is asleep the puncture is made with the stylette of the needle removed. Once a satisfactory flow of blood is obtained the needle is secured firmly with the

FIG. 1. Cinefluorographic apparatus, showing position of patient.

fingers of one hand while the stylette is replaced with the other hand. A few seconds are then required for the radiologist to set the machine for filming. As soon as he is ready the stylette is removed and the syringe with dye is attached to the needle, making certain that a free flow of arterial blood is still occurring. At a prearranged signal the injection is started rapidly and the exposure is begun at the same moment. This insures filming the beginning of the ascent of dye up the internal carotid. The exposure is continued for a total of not more than 5 sec. Usually $3\frac{1}{2}$ sec. is ample to visualize the whole of the arterial phase. During a single such 5 sec. exposure the patient receives a dose of about 25 r of radiation. The operator's hands are kept out of the field by the use of a cone reaching from the tube nearly to the patient's head. A leaded shield can be conveniently interposed between the patient's chin and the operator's hands if desired, although one is safe without it.

We have employed diodrast varying in concentration from 35 to 70 per cent, but are most satisfied with 50 per cent. Legible films are obtained, however, with 35 per cent. The test is not done if the patient is found sensitive to either of the two preliminary diodrast sensitivity tests, the intradermal or the intravenous (5 cc.), both of which are done routinely. Satisfactory records have been obtained

with as little as 6 cc. Eight cc. are used as a rule. As soon as the injection of dye is made the needle is withdrawn and pressure applied over the puncture site for a few moments. The entire procedure from the moment of the patient entering the room until leaving has frequently been done in 20 min.

For an immediate reading one can inspect the wet film either directly or with a magnifying glass, or a single frame can be projected on a screen if so desired. It requires 12 min. to develop the film. Thirty-five mm. film is used and since projection is more convenient with a 16 mm. projector we generally prefer waiting a day until the reel is optically printed on 16 mm. film. It is best studied then by splicing both ends of the film together to form a loop and running it through the projector as long as desired. For more detailed study in slow motion, frames may be duplicated to as high a ratio as 3 frames to 1 to obtain a comfortably slow speed without flicker.

FIG. 2. Frames from a normal cinefluorographic cerebral angiogram.

The cinefluorographic unit consists of a 35 mm. camera equipped with a f/0.85 lens and a DuPont-Patterson E-2 screen. The roentgen-ray generator is the KX-1 and the roentgen-ray tube is the CRT-1-2, both manufactured by the General Electric X-Ray Corporation. The film used is 35 mm. Linograph Ortho. The camera

speed is 60 frames per sec. which results in moderate slow motion when projected. The camera is operated synchronously with the 60 cycle alternating current, and by resorting to half-wave rectification the roentgen-ray impulses which would ordinarily occur during the shutter-closed phase of the camera are suppressed. This results in 60 exposures per sec., each exposure lasting about 1/246 sec. One hundred kV. and 150 mA. are constant factors for all exposures. At 100 kV. the tube with an added filter of 1 mm. of aluminum gives a half value layer of 2 mm. of aluminum and an estimated threshold erythema of 350 r. The target-screen distance varies between

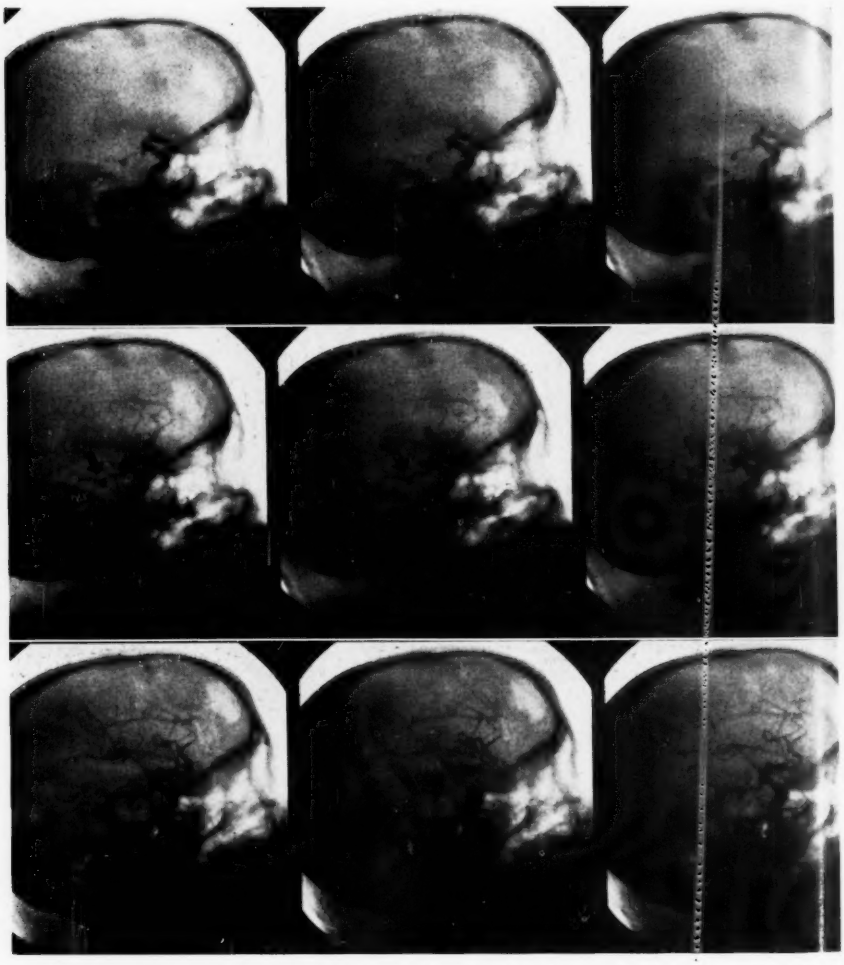

FIG. 3. Frames from a normal cinefluorographic cerebral angiogram. Arrows point to a collection of dye which until resolved by rotation of the head later in the angiogram might have been mistaken for a berry aneurysm. See text.

FIG. 4. Frames from a cinefluorographic cerebral angiogram in a patient with occlusion of the internal carotid artery at the bifurcation of the common carotid, confirmed at operation. Arrows point to head of column of dye ascending the common carotid. Further in the record branches of the external carotid fill.

28 and 30 in., resulting in a total dose to the patient of between 20 and 25 r. An adjustable lead diaphragm is used to narrow the roentgen-ray beam to the area under observation.

We have employed the same method for vertebral artery angiograms with success according to the technique of Sugar, Holden and Powell.[3]

In a recent instance during the injection the patient rolled his head freely. Instead of distorting the vascular picture as was anticipated, it had the effect, when projected, of providing an impression of a three-dimensional image as

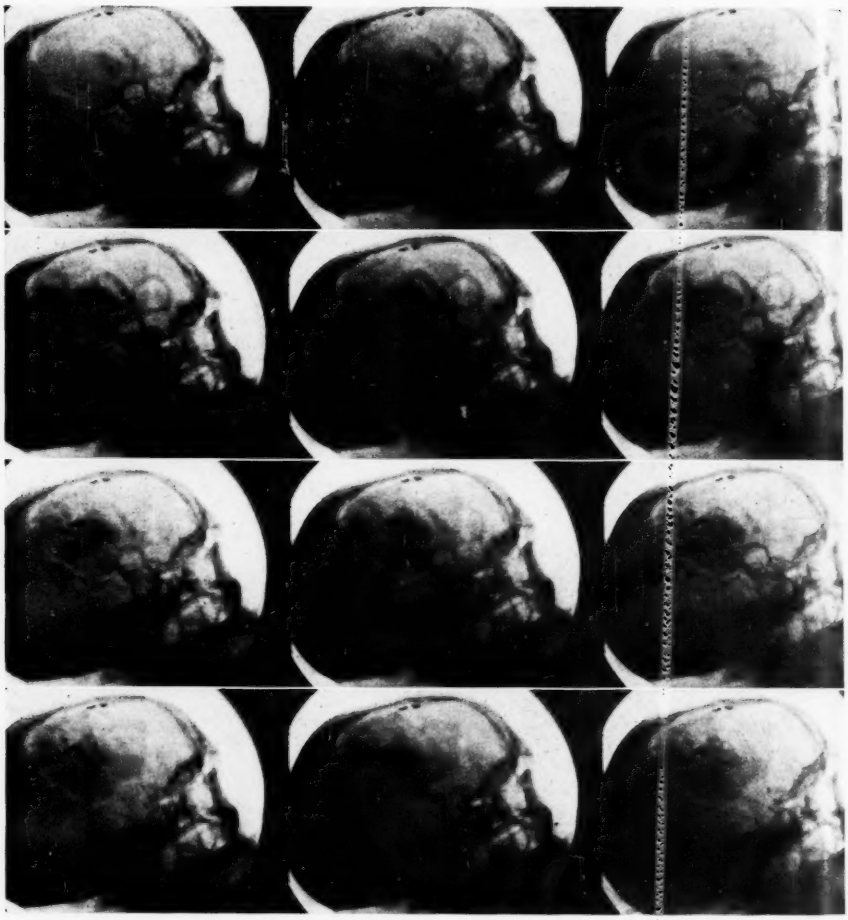

FIG. 5. Frames from a cinefluorographic cerebral angiogram in a patient with an arteriovenous vascular malformation, postoperative. There is venous filling in the last two strips.

the vessels were thrown into different orientations. We hope to utilize this feature deliberately in the future. In the above instance a small concentration of dye, which might have been taken to be a berry type of aneurysm at the junction of the posterior communicating artery with the posterior cerebral (Fig. 3B), was shown clearly by this rotation to be only the end-on view of these vessels overlapped.

Except in pathological conditions in which a prominent venous phase occurred, as in an arteriovenous malformation, we have been unable thus far to adequately visualize the venous phase of the injection consistently.

Figs. 2, 3, 4 and 5 show a few frames in different phases in each of four different angiograms. Progression of the dye from one frame to the next is

not recognizable in still pictures but can be made out from one strip of frames to the next.

From our experience thus far, we feel that the cinefluorographic method has certain advantages over the conventional methods of cerebral angiography. Requiring only a single small injection as opposed to the multiple larger ones usually required to insure satisfactory timing of exposure or for stereoscopic views in still angiograms, it is a much easier, more certain and less traumatic procedure to perform. Offering a continuous record as it does, there is no chance to miss an essential phase of the arteriogram as sometimes happens with still angiograms. The failure of a certain vascular trunk to fill can be accepted with certainty as evidence of occlusion of that vessel, whereas with single exposures one might not know whether an exposure a fraction of a second later might not have shown that vessel filled. The possibilities of obtaining useful information from visualization of feeding vessels to tumors or vascular malformations or to aneurysms remain to be explored. The investigation of cerebral circulation, normal and pathological, may be facilitated by the use of this method.

In some respects, however, this technique in its present form has certain shortcomings. Perhaps the major one is that at its best the quality of the image lacks the sharpness and definition of good still angiograms. The legibility, however, is still wholly adequate for clinical purposes, and the films are easily and comfortably read. The lack of stereoscopic views is not a serious deficiency inasmuch as the kinetic record plus the rotational feature previously mentioned promises to enable the reader to localize the depth of the vascular image without difficulty. We have not attempted anteroposterior views. The poor quality of the venographic phase may yet be overcome and it appears that if it is prominent enough to visualize pathologically this technique will record it.

SUMMARY

A method is described for taking cinefluorographic cerebral arteriograms which are suitable for clinical use. This depends upon the availability of a practical cinefluorographic apparatus such as has been constructed at Strong Memorial Hospital.

The advantages of this method over conventional still angiography have been pointed out. The greater simplicity, facility and potential yield of information of this method seem to outweigh the disadvantage of the somewhat inferior quality of the image when compared to good still angiograms.

We are indebted to the following persons without whose assistance and guidance this work would not have been possible: Dr. J. S. Watson, Jr., Dr. G. H. Ramsey, Dr. William P. Van Wagenen, and Dr. William S. McCann.

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HISTOLOGIC RECOGNITION OF SYMPATHETIC TISSUE

JOHN THEODORE ROBSON, M.D.*
Tacoma, Washington

(Received for publication August 4, 1949)

IN THE course of surgery of the sympathetic nervous system it is frequently of value histologically to identify tissue resected, both as a guide to the operator at the time of surgery and as a matter of record. Histologic differences exist between cerebrospinal peripheral nervous tissue and sympathetic tissue, but these are mainly differences in size of axon and degree of myelination. Sometimes the pathologist can report resected tissue only as "nerve" or "nerve ganglia." In cases where dorsal root ganglia are resected along with sympathetic ganglia, as in sympathetic denervation for angina or vascular disease of the upper extremity, it is often frankly impossible to distinguish the ganglia histologically. With this problem in mind, we have recently worked out a satisfactory method of histologic identification based on differences in the Schwann cell of sympathetic and cerebrospinal nerves.

Key and Retzius[3] in 1873 were the first to describe the Schwann cell, although Adamkiewicz[1] published an independent description later. The existence of these cells was at first disputed and Rosenheim,[7] in particular, thought them mast cells. Reich[5] studied this problem and distinguished the two cells by chemical and staining differences of the granules in the cytoplasm. He noted that the granular matter in the cytoplasm of the Schwann cells could be extracted by alcohol warmed to 45° and not by cold alcohol, and that it was various shades of red[2] when stained with metachromatic basic aniline dyes such as polychrome methylene blue, thionine, toluidine blue, and safranine. In this manner they resembled the lipid matter of the central nervous system that Liebreich[4,8] had named protagon, and so became known as protagon granules. This was later shortened to the Greek letter pi (π) by analogy to the Greek letter mu (μ) used for Elzholz bodies of peripheral nerves.

Investigation[6] on specimens of peripheral nerves from 266 patients has shown these structures to be present in every section taken from individuals 8 years old and older, and never present below this age. Sections taken through the spinal cord in such a manner that the anterior and posterior nerve roots are present show that the granules begin to appear just distal to the glial cone where the axons become invested with neurolemma. Immediately adjacent to the glial cone, however, the Schwann cells appear to be in transition with the oligodendroglia, and protagon granules are not present. All other Schwann cells distally, however, throughout the peripheral nerve, exhibit these structures, except that the Schwann cells of the dorsal root ganglia immediately adjacent to the ganglion capsular cells, and in transition with the capsular cells, do not contain protagon granules.

* Medical Arts Building, Tacoma 2, Washington.

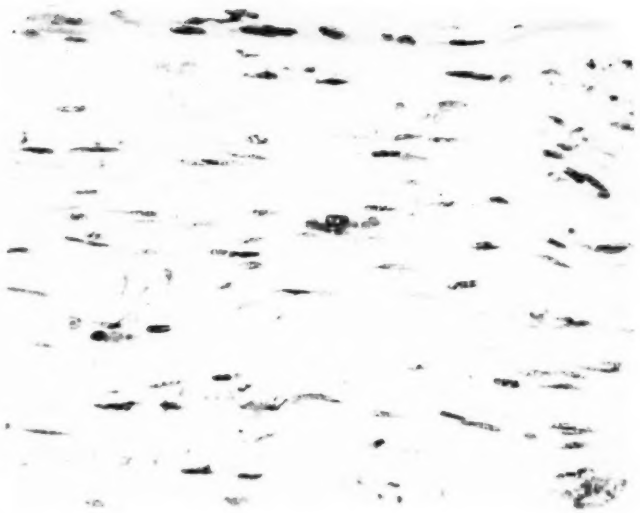

FIG. 1. Longitudinal section of a fasciculus of the sciatic nerve. The protagon granules are seen at the poles of the nuclei of several Schwann cells. Cresyl violet stain, ×275.

FIG. 2. Greater splanchnic nerve at point of entrance to coeliac ganglion. A group of medullated fibers are seen to the left of the midline with one Schwann cell present containing protagon granules. Cresyl violet stain, ×275.

Protagon granules of the sympathetic nervous system were studied by examining 55 specimens of the supra- and infra-diaphragmatic portion of the greater splanchnic nerve with a portion of the coeliac ganglion attached. This portion of the sympathetic nervous system was chosen because it contains all three types of sympathetic (orthosympathetic) fibers. The greater splanchnic nerve is largely composed of visceral afferent and preganglionic visceral efferent fibers, but the splanchnic ganglion present in the course of the nerve, opposite the 11th or 12th thoracic vertebrae, contributes postganglionic visceral efferent fibers.

It was found that the Schwann cells of the sympathetic nervous system differ from Schwann cells elsewhere in that they are without protagon granules, except for those found on an exceedingly few heavily myelinated axons. These fibers seem too few to fall into the groups of recognized sympathetic fiber types. In any event, the extreme scarcity of protagon granules is so marked that a glance is sufficient to distinguish sympathetic from cerebrospinal nervous tissue.

It was found that the most satisfactory specific stain for these granules was cresyl violet, but with experience the hematoxylin eosin stain will suffice. With cresyl violet stain the granules are seen as a brick red against a blue background. With hematoxylin eosin stain the granules are faintly discernible as granular matter in the Schwann cell cytoplasm.

SUMMARY

Protagon granules are uniformly present in the Schwann cells of the cerebrospinal nerves of individuals 8 years old and older. Only an exceedingly few Schwann cells of the sympathetic nervous system contain these granules. By utilizing this difference sympathetic and cerebrospinal nervous tissue may be readily distinguished.

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CLOVIS VINCENT

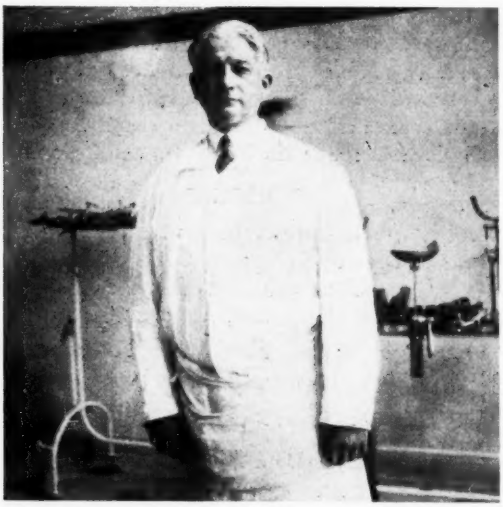
HIS LIFE AND MAIN CONTRIBUTIONS TO NEUROSURGERY

JACQUES LE BEAU, M.D.,* AND J. B. TAVERNIER, M.D.
Paris, France

(Received for publication December 19, 1949)

HIS LIFE

CLOVIS VINCENT was the son and grandson of country doctors. He was born at Ingré, a few miles north of Orléans, on September 26, 1879, and died in Paris, first Professor of Neurosurgery, on November 14, 1947. He was an exceptional individual in every respect, animated with an inspiring passion in his search for progress in science, and surrounded by utterly devoted pupils and friends. As happens to any great man, he made



Clovis Vincent in his operating room at *la Pitié* in 1933.

some enemies. But it is not our purpose to try to describe the unselfish tenacity thanks to which he created French neurosurgery: nowhere can a better description of it be found than in his biography by his friend Professor Réné Moreau,[17] and in his own famous inaugural address delivered in 1939.[29] He was *par excellence* the man to whom should be applied the very words he used there to describe Cushing: "He interests himself only in the cure of the patients and the advance of science." Add to that natural charm, some shyness, a never failing kindness of heart, and Clovis Vincent's outstanding

* Neuro-chirurgien des Hôpitaux de Paris, Paris, France. Address: 107, Rue de l'Université, Paris 7e, France.

prestige will be understood. Physically he was an athlete with a passion for arduous sports; he was absolutely fearless and in the first world war promptly became as famous among the soldiers as he already was in peace time among the neurologists. During the second world war his life was described in this *Journal* by his pupil Rabinovitch.[19]

Vincent's scientific life from 1905 to 1926 was devoted essentially to neurology. Still very young he met Babinski, understood at once the greatness of this difficult and silent master, and followed his teaching with the same patience and insight during his whole career. There is no doubt, as Vincent used to say, that the precise knowledge of neurology he thus acquired was decisive in allowing him to be not only a neurosurgeon, but a neurosurgeon who contributed to the advance of brain physiology. Vincent was then *Médecin des Hôpitaux de Paris* at Tenon, and not a surgeon.

Since 1926 at least, as one of us who had the privilege of being Vincent's friend for 30 years can testify, Clovis Vincent had wanted to create real neurosurgery in France. In 1927 he went to the United States and spent less than a month with Harvey Cushing. There was the same understanding as with Babinski, but here language differences were somewhat of an obstacle. However, Vincent observed every single gesture of Cushing, saw their meaning, and fully appreciated the greatness of his new master. Back to Paris, surmounting innumerable difficulties, he started modern neurosurgery, first with de Martel, and later alone. His outstanding accomplishments led to the unprecedented event of building surgical theatres for him in his medical service at *la Pitié* in 1933. In 1938, with the help of the Rockefeller Foundation, a chair of neurosurgery was founded in Paris, to which Vincent was the first to be appointed. Now, in his turn, he was held as a master all over the world, and, what counted more, Vincent's admiration for Cushing was then reciprocated, as has been told in Fulton's Biography[7] and in Jefferson's article.[9] To Clovis Vincent, the words that Cushing used were the greatest praise that one could wish for.

In December 1948, about a year after Vincent's death, the Société de Neurochirurgie de Langue Française was founded in Paris, with Prof. Petit-Dutaillis as President. No such thing would have been possible without Clovis Vincent's achievements. In March 1948, neurosurgery, already established at the Faculté de Médecine by the founding of a chair, was officially recognised by the Hôpitaux de Paris in the creation of *Neurochirurgiens des Hôpitaux de Paris*, and three of the four nominated were the first three surgical pupils of Clovis Vincent. The tribute of French neurosurgery to Vincent will be endless.

HIS CONTRIBUTIONS

We would like to mention as briefly as possible the main contributions of Clovis Vincent in the field of neurosurgery. Some of them are well known abroad, many others are not. It is not possible to include here a complete list of references to his works. They comprise not only his own many publica-

tions, but also numerous articles and theses directly inspired by him, all of which came from the Hôpital de la Pitié, a few, indeed, having been published even after his death. In analysing Vincent's works we have grouped them as follows.

Neurology. For twenty years, Clovis Vincent was the leading neurologist of Babinski's school, and his scientific publications from 1908 to 1928, even during the 1914–1918 war, were numerous and valuable. He discovered albuminuria in cases of meningeal diffuse hemorrhages, and new aspects of serous disease. He studied traumatic shock. Later he devoted himself to von Economo's disease. Several of his best known publications are on hysteric phenomena and similar but *organic* syndromes.[21] One of his last purely neurological contributions is on the treatment, by blood transfusion and cardiotonic drugs, of hemiplegia following loss of blood and fall of blood pressure in arteriosclerotic patients.[44]

General Neurosurgical Problems. Pneumoventriculography and pneumoencephalography were studied at length by Clovis Vincent and his school in the years 1928–1934. Other general problems were also investigated and particular attention may be called to his publications on ventral decubitus to combat postoperative acute lung oedema.[30] Ventral decubitus is also a life-saving procedure for patients from whom a large cerebellar or acoustic or fourth ventricle tumor has just been removed: in such instances Vincent pointed out that it prevents the medulla and pons from falling backwards and lessens the accidents of ventricular depletion.[15] He also described the treatment of vascular collapse following the removal of certain tumors.[36]

Finally it must be said that during his neurosurgical career, Vincent interested himself above all in brain stem disturbances. Though he never published a special work on this problem, he frequently alluded to it, sometimes in detail, in many of his articles,[23] especially in those on head injuries. His main teaching was to emphasize the importance of careful observation for signs revealing disturbance of the brain stem at its very onset, since they are too often the forerunners of a fatal evolution of the disease. Such are disorders of deglutition, acceleration of respiration, hyperthermia, vasomotor disturbances of body and face, stiffness of neck, and blurring of consciousness. All of them, easy to show at a late phase, must be sought for at an early stage, and this is where Clovis Vincent exhibited incomparable patience, observation and flair.

Brain Oedema—Temporal Herniation—Cerebellar Herniations. To these three closely allied questions, Clovis Vincent devoted perhaps the greater part of his time. From the beginning to the end of his neurosurgical life he studied generalised brain oedema, trying to find the cause and to fight against it.[10] He actually discovered acute brain oedema, one of the most extraordinary phenomena met with in physiology.[24] He described its two forms: the frontal, sometimes curable, and the cerebellar, always fatal when accompanied by a sharp increase in blood pressure.[27] Physiological and clinical studies continued to the present time.[12]

Temporal herniation, frequently present with brain oedema, though previously mentioned from an anatomical standpoint, actually was explained by Clovis Vincent as far back as 1930.[40] Since that time his school repeatedly studied the phenomenon, its causes, its signs, and its possible cure. Temporal herniation may be the most important cause of death in cases of brain tumor[35] and abscess, and it is a factor in head injuries also; its main signs are impairment of consciousness and stiffness of the neck. Temporal herniation may play a part in the production of generalised brain oedema; in any event, its pressure on the brain stem is the explanation of certain signs of supratentorial tumors. All of these points were fully recognised and published by Vincent in the 1930's.

Lastly it was under Clovis Vincent's direct leadership that a special complication of cerebellar oedema was discovered: herniation of the cerebellar roof above the tentorium, which could lead to diagnostic errors with tumors of the posterior part of the third ventricle.[38]

Brain Tumors. All the early neurosurgical works of Clovis Vincent are on the subject of brain tumors, among which was a report in 1929 of his operative results in a group of cases.[34] After the development of neurosurgery at *la Pitié* in 1933, about 500 brain tumors were operated on every year. It would be redundant to enumerate the articles published from *la Pitié*. Among the most significant were those on sphenoidal wing[5] and suprasellar meningiomas,[8] craniopharyngiomas,[6] acoustic tumors,[4] cerebellar hemangiomas,[20] corpus callosum gliomas,[43] and epilepsy.[16] They were all inspired by Clovis Vincent and, when published under another name, were even partly written by him.

Let us however single out two works: one is the monumental thesis of our late friend and colleague Guy Loisel on cerebellar astrocytomas.[14] (Loisel, one of the favorite pupils of Vincent, died in 1940.) The other is the outstanding report written by Vincent on frontal lobe tumors;[22] published in 1928, it is a model of clinical analysis and insight; it introduced in France the new neurology born and bred in neurosurgery.

Brain Abscess—Suppurative Meningitis. Before the introduction of penicillin, Vincent's method for the cure of brain abscess established certain important steps in the management of this difficult problem. It consists of two stages: (1) Decompressive flap, in front of the abscess without opening the dura, to fight against brain oedema and pressure on the brain stem by temporal herniation. (2) Two or 3 weeks later, excision in one piece of the abscess, then encapsulated as successive tappings show it to become.[25]

This now classical method is known all over the world and we shall not dwell at length upon it, since it is perhaps the main reason for Clovis Vincent's fame abroad. Let us say, however, that brain abscess was always a favourite theme of Vincent, and after 1944, when penicillin was available to us, he directed subsequent work to extend his radical excision method to the field of multilocular, metastatic, and meningeal abscess.[11] He personally published the results of treatment of suppurative meningitis with penicillin.[41]

Tuberculomas—Tuberculous Meningitis. Tuberculomas of the brain were much more common in France than in the United States, for example, and their surgical cure was a major problem. Clovis Vincent held the following principles to excise them successfully, that is, without postoperative tuberculous meningitis.[33]

(1) The correct diagnosis should be at least strongly suspected before opening the dura. That depends mainly on very thorough clinical information. Careful exploration with a Cushing's large blunt needle is imperative, looking for the peculiar hardness *en masse* of the tuberculomas, which differs from the resistance of an abscess shell, and is more stone-like than a meningioma.

(2) The tuberculomas should be unique with no or few adhesions to the dura.

(3) Excision has to be made in apparently normal brain tissue, without opening the tuberculomas, and with the diathermic forceps.

(4) Postoperative meningitis comes not only from the direct communication of the tuberculomas with the subarachnoidal spaces, but from contamination through the blood running over the lesion and soiling the meninges. It is therefore necessary to operate under constant protection with Zenker impregnated swabs, which must be put at once into every new brain wound during the whole operation.

When Clovis Vincent's method was applied, 90 per cent of the tuberculomas were cured; of these, 70 per cent were cerebellar tuberculomas, the mortality in the latter group having been 95 per cent previously.

Finally, after the introduction of streptomycin, a new neurosurgical approach was directed against tuberculous meningitis with, of course, inconclusive results as yet.

"Inflammatory" Diseases of Brain and Meninges. By this expression, Clovis Vincent meant the extensive group of "pseudo tumors," on which he laid particular emphasis. First of all, he interested himself in the pseudo pituitary tumors, and conceived the idea of a surgical entity that he called *opto-chiasmatic arachnoiditis*,[3] one of the most frequent causes of classical retrobulbar neuritis. Clinically, impairment of vision is progressively very marked, central scotoma is common, and the discs are either normal, or pale, or pink and slightly blurred. The sella turcica and ventricles are radiologically normal. At operation several anatomical types are met:[39]

(1) Cystic arachnoiditis, with definite compression of optic nerves by the arachnoidal cyst and adhesions; usually here postoperative improvement is quickly achieved.

(2) Fibrous arachnoiditis, where both optic nerves (or one) are stuck in a fibrous sheath and exhibit a variable degree of atrophy; here the prognosis is not so good.

(3) A form where the cisterna anterior is practically normal, but frontal lobe retraction seems to play a part in the postoperative visual improvement, which is a common occurrence. This type may depend on a special kind of

encephalitis, as is indicated by the occasional coincidence of epileptic fits, and of chronic hypothalamic disorders.

In a few instances the opto-chiasmatic arachnoiditis was of syphilitic origin; in most cases there was no precise cause (some may have been vascular); hundreds of them were operated on at *la Pitié*.

Clovis Vincent was very keen too on cisterna magna arachnoiditis, usually cystic and simulating a cerebellar or 4th ventricle tumor. It is a frequent cause of subacute hydrocephaly of adults.[2] Finally he described the pseudo tumoral encephalitis, sometimes with choked discs, either generalised (the so-called *gros cerveau*), or localised, for instance, to a cerebellar hemisphere or to a temporal lobe.

Vascular Intracranial Disorders. The main interest of Vincent in this category was directed towards the early diagnosis of angiomas and aneurysms of the brain. He insisted upon the strong suspicion of such a condition in patients suffering from repeated diffuse meningeal hemorrhages.[37] Other vascular disorders especially studied in *la Pitié* were the intracranial form of hypertensive disease, intraventricular hemorrhage,[18] and the non-traumatic hematomas, either subdural, or intracerebral, or intracerebellar.

Head Injuries. Clovis Vincent adopted a new line of approach to the management of head injuries. He compared them to the "measured injury," as he called it, suffered by patients after an intracranial operation. In other words, he started by carefully examining what he found upon reopening the operative wound in those cases where the patient's condition necessitated a secondary procedure.

Thus he came to the conclusion that in head injuries the main signs indicating that operation should be performed are the signs of pressure on or a lesion of the brain stem (described above). Alteration of the brain stem is much more important than alteration of the cerebral hemispheres as far as the vital prognosis is concerned. Vincent laid special emphasis on the study of deglutition in head injuries as a factor in prognosis.

As to the diagnosis of the variety of intracranial disorder requiring operation, Clovis Vincent, in spite of his primarily neurologic education, came to the definite conclusion that neurological examination alone is generally insufficient. He advocated strongly the method of exploratory burr holes to look "at" the dura and cortex and "for" the ventricles. It is the only way to distinguish the hematomas (extradural, subdural, intracerebral), brain oedema, serous meningitis, internal hydrocephaly, or those rare but real states of intracranial hypotension (either cerebral collapse or ventricular hypotension or both).[26,28]

For open cranio-cerebral wounds, at the very beginning of the war, he recommended with much energy a large approach, with clean excision of the brain wound, careful hemostasis, and closure of the dura.[31,32]

Surgery of Pain. The most original researches of Vincent in surgery of pain were in two fields. The first is the section of the spinothalamic tracts in the medulla (unpublished). The second is the section of the trigemino-

thalamic tract, or, to be more specific, of the quintothalamic tract, at the pontic level, against heterolateral pain, mostly postherpetic. This work, including eight observations, was published after Vincent's death, but all operations had been performed by him.[13] He had also started some more recent work on the treatment of intractable pain by frontal lobe surgery.

Surgery of Spinal Cord. Clovis Vincent devoted himself for a long time to the clinical diagnosis between spinal cord tumors and other conditions, mainly Pott's disease; he insisted mostly on the differences in the muscular perivertebral rigidity. Later he analysed and improved in some respects the Queckenstedt test, making it more sensitive after withdrawal of a few cc. of cerebrospinal fluid.[42] Later again, vertebral and spinal angiomas were described at *la Pitié*.

Such are Clovis Vincent's main contributions to neurosurgery. Let us say that he was most keen on brain oedema, temporal herniation, brain abscess, tuberculomas, opto-chiasmatic arachnoiditis, and brain stem disturbances in head injuries. In all these problems he led science to advances, and he will not be forgotten, either in France or among neurosurgeons abroad.

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CASE REPORTS AND TECHNICAL NOTES

UNILATERAL VERTEBRAL ARTERY LIGATION

REPORT OF A CASE ENDING FATALLY WITH THROMBOSIS OF THE BASILAR ARTERY

LYLE A. FRENCH, M.D., AND GERALD L. HAINES, M. D.
Division of Neurosurgery, University of Minnesota Hospitals, Minneapolis, Minnesota

(Received for publication June 11, 1949)

Ligation of the large vessels of the neck has always been viewed with some hesitancy by surgeons because of the ever present danger of brain ischemia. In the 151 odd years since Abernathy[1] ligated the common carotid artery for a lacerated internal carotid, confidence in this procedure has increased until now such ligations are rather commonplace. Dandy[2] stated that immediate complications are less than 4 per cent and delayed, less than 2 per cent. However, Reid and Andrus (cited by Keegan[5]) believed that the hazard of ischemia is 20–25 per cent.

Ligation of the vertebral arteries has been relatively rarely performed. Matas[6] stated that Maisonneuve and Favrot performed the first ligation in 1852, for a gunshot wound, thus disputing the statement of Sanson, made in 1836, that the "wounds of this vessel are beyond the resources of art." Ligations of the vertebral vessels had increased in frequency to a point where Dandy[2] stated, based on the work of Alexander, that "both vertebrals can be ligated, even at the same operation, with impunity."

With the increasing popularity of the angiogram as a diagnostic procedure, and thus the frequent exposure of the great vessels, and with increasing knowledge of the value of ligation in the treatment of vascular lesions, it has become increasingly important to know accurately the risk involved with ligation. It is our purpose here to report a case of unilateral vertebral artery ligation, done following an angiogram, resulting in brain stem ischemia and death.

CASE REPORT

A 48-year-old white male was admitted to the University of Minnesota Hospitals on Dec. 9, 1948 with a history of progressive neurological deficit of 11 years' duration.

There had been 3 previous admissions, the first of which was on Aug. 5, 1939, at which time a history was obtained of dizzy spells, occipital headaches, visual hallucinations, and gait disturbance. Examination revealed a right facial paresis of central type, bilaterally positive Hoffmann signs, decreased left abdominal reflexes, and disturbance of equilibrium. CSF protein was 90 mg. per cent. Skull roentgenograms revealed no abnormality. He was discharged, but kept under observation as a brain tumor suspect.

Because of persistent neurological abnormalities, he was readmitted on Mar. 8, 1940. At this time, all his deep tendon reflexes were hyperactive, the Hoffmann sign was positive on the left, the right abdominal reflex was reduced and the speech slurred. A ventriculogram revealed a defect in the occipital horn of the left lateral ventricle. A left occipital craniotomy was performed and a very vascular fibroblastic meningioma arising from the upper surface of the tentorium was encountered. Subtotal removal of the tumor was done, total extirpation being impossible because of extreme vascularity. A right homonymous hemianopsia was present after operation.

The patient was readmitted on May 16, 1940. He was feeling well, but there was some recent memory loss, a mild expressive aphasia, and a right homonymous hemianopsia. The

craniotomy wound was reopened but, in spite of ligation of the left lateral sinus and multiple transfusions, total removal of the tumor was not accomplished because of excessive bleeding.

Examination on Final Admission, Dec. 9, 1948. The patient was more lethargic and profound personality changes had developed. Psychological testing revealed marked mental deterioration. He had had numerous generalized convulsive seizures. There was right facial paresis of central type, a positive left Hoffmann sign, left dysdiadochokinesia, trunkal ataxia, questionably positive Romberg sign, right homonymous hemianopsia, and a formulation aphasia with anomia and alexia.

Operation. A further attempt at removal of the tumor was considered. It was deemed advisable to first ascertain the source of blood supply to the tumor so that the extreme vascularity could be overcome at the onset of the operation. A vertebral angiogram (Fig. 1) was made by the open method, the left vertebral artery being visualized some 2 cm. above its origin from the subclavian artery. It was about the size of the usual common carotid artery. The angiogram revealed a large mass of small vessels, occupying an area about 8 cm. in diameter in the left cerebellar region and extending well above the tentorium. The source of blood to the tumor was obviously the posterior cerebral arteries. In order to reduce the supply, the left vertebral artery was ligated.

FIG. 1. Vertebral arteriogram. Meningioma arising from tentorium with blood supply arising from right and left posterior cerebral arteries.

Course. On the evening of the 1st postoperative day it was noted that the patient had a left Horner's syndrome, horizontal nystagmus of the left eye on left lateral gaze, and paralysis of medial gaze in the right eye. Aphasia had increased. By the next day there was right hemihypesthesia and hemiparesis and an increase in the right facial paresis. In the left eye the quick component of the nystagmus was to the left on lateral gaze and rotatory on upward gaze. Deep reflexes on the right were increased and there were positive Mayer, Oppenheim, and Babinski signs on the left. On the 3rd day he was somewhat improved; the right ophthalmoplegia had largely disappeared. However, during the night he was observed to cough and choke while taking medication and following this he became stuporous and expired.

Autopsy. There was thrombosis of the entire left vertebral artery and of the basilar artery up to $2\frac{1}{2}$ cm. above the origin of the superior cerebellar artery. The pontine tissue on either side of the upper portion of the basilar artery was soft, the softening extending downward more on the left. In the left parieto-occipital area and involving the greater part of the left cerebellar hemisphere was a firm, reddish, nodular tumor mass, some $6 \times 5 \times 4$ cm., appearing to arise from the tentorium (Fig. 2). Histological diagnosis: Fibroblastic meningioma. The cause of death was considered due to infarction of the pons.

DISCUSSION

Ligation of a vertebral artery has been accomplished without complications. Shumacker[8] ligated the vertebral artery in 4 cases of traumatic arteriovenous fistulae, in 1 case one vertebral and one common carotid, without ill result. However, he did mention 1 other case in his experience in which there was homolateral cerebellar necrosis occurring without evidence of embolism or thrombosis. Elkin and Harris[3]

reported 10 cases, 1 bilateral, of vertebral ligation, the only ill effect being a Horner's syndrome in 1 case. Dandy[2] stated that one vertebral artery had been ligated between the atlas and the occipital bone "perhaps twenty times" without ill result. He also reported a death following a two-second compression of one vertebral when the other had been ligated.

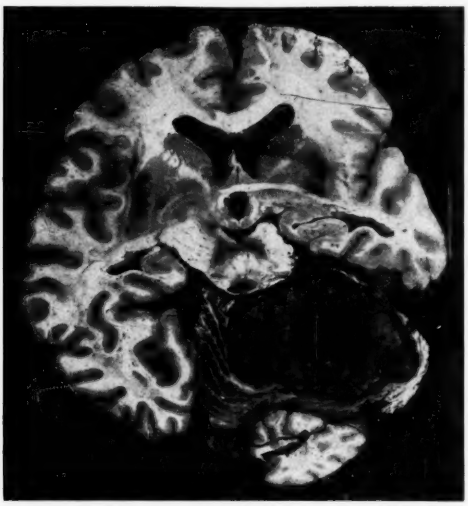

FIG. 2. Coronal section revealing large meningioma displacing cerebrum and cerebellum.

The etiology of the ischemia following vertebral or carotid artery ligation is not yet clear. In the case of the carotid artery, Olivecrona[7] reviewed the literature and concluded that the determining factor is pre-existing insufficiency of the cerebral circulation caused by such factors as changes in pressure, hemorrhage, or infection. He was of the opinion that thrombosis in itself rarely produces ischemia, and that in the cases that end fatally, thrombosis is rarely found. Dandy[2] believed that thrombosis and embolism are of extreme importance. Holman[4] commented on the advantages of fractional ligation. The final answer seems yet to come, as the important problem of vascular stasis remains nearly untouched. In our case it seems probable that there was intimal damage at the time of ligation with resulting thrombosis, but that in addition, the enlarged vertebral artery was carrying an unusually large proportion of the total intracranial blood flow, which, when cut off, produced fatal ischemia.

SUMMARY

A case of unilateral vertebral artery ligation with the purpose of reducing the blood supply to a vascular tumor, but resulting in brain stem necrosis and death, is reported. Pertinent literature is briefly discussed. It is apparent that such ligations are not without their perils.

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METASTATIC PINEAL TUMORS

A CLINICOPATHOLOGIC REPORT OF TWO CASES*

VICTOR N. TOMPKINS, M.D., WEBB HAYMAKER, M.D., AND ELDRIDGE H. CAMPBELL, M.D.
The New York Division of Laboratories and Research, Albany, New York, The Army Institute of Pathology, Washington, D.C., and the Department of Surgery, Albany Medical College, Albany, New York

(Received for publication July 11, 1949)

Metastasis to the lung from pineal tumor is known to be exceedingly rare. However, among 13 fatal cases of pineal tumor which reached the Army Institute of Pathology during World War II there were 2 in which metastasis to the lung had occurred. Only 1 such case has been previously reported—that of Stowell, Sachs and Russell.[25] Their patient, a boy of 15, had been ill for 3 weeks before entering hospital. At operation a large tumor, diagnosed as a primary intracranial chorionepithelioma, was removed from the pineal region. Death occurred approximately 3 months after the onset of symptoms. On postmortem examination no pineal tissue was found, but there were many arenaceous bodies in the region where the pineal body normally is present. The conclusion reached was that the tumor arose as a teratoma in the pineal region and that its chorionepitheliomatous portion grew rapidly at the expense of the other elements, destroying the original teratoma and the pineal body. The tumor had invaded the superior sagittal sinus and other blood vessels, and had metastasized to all lobes of both lungs.

Metastasis of other primary intracranial tumors to extracranial structures is also infrequent. This mode of spread has usually occurred by way of the blood stream or lymphatics, or both, as will be noted in the following examples gathered from the literature: basophil adenocarcinoma of the hypophysis with metastases to liver;[4] chromophobe cell carcinoma of the hypophysis to liver;[18] adenocarcinoma of the hypophysis to liver and hepatic lymph nodes;[8] adenocarcinoma of the hypophysis to liver, kidneys, urinary bladder, uterus, vagina and para-aortic lymph nodes;[11] benign adenoma of the hypophysis to bones;[27] malignant mixed chromophobe and chromophile hypophysial tumor to cervical lymph nodes;[9] large-cell carcinoma of the hypophysis to cervical lymph nodes, lungs and pleura;[3] sarcomatous meningioma to cervical lymph nodes;[12] meningioma to one of both lungs;[5,6,15,16,28] meningioma, in 3 cases, 1) to lungs, pleura, abdominal lymph nodes, lumbar vertebra, 2) to lung, liver, mediastinum, and 3) to pleura;[21] hemangioblastoma to lungs and hilus nodes;[1] medulloblastoma to subcutaneous tissue over sternum;[23] medulloblastoma to vertebrae;[19] spongioblastoma multiforme to lung, shoulder region and arm;[7] glioblastoma multiforme to lungs;[17] astroblastoma to thoracic vertebrae, ribs, lungs and peribronchial lymph nodes;[24] probable atypical oligodendroglioma to neck.[26]

CASE REPORTS

Case 1.† A white male, aged 34 years, entered hospital‡ on Feb. 1, 1944. For 6 weeks he had had dull, constant headache, bifrontal and left occipital, and had noted visual fatigue on reading. For 5 weeks he had been unable to write, though he understood what he read and knew what he wanted to write. He denied having had motor weakness

* This study supported in part by a grant from The American Cancer Society to the American Registry of Pathology.

† Army Institute of Pathology Accession 157997.

‡ 33rd General Hospital, then in Tunisia.

or altered sensation. It is noteworthy that he had served as an infantry squad leader in combat until 3 weeks before admission.

Examination. The patient could understand adequately the spoken and written word; however, his writing was poorly performed. While walking, he held his head stiffly erect and whereas the left arm was kept partly flexed, the right hung immobile. The Romberg test was positive, the patient tending to fall backward and slightly to the right. Coordination and succession tests were normal on the left side of the body but were defective on the right. There was motor weakness of the entire right side, including the lower part of the face. The right palpebral fissure was narrowed. On the right side the deep reflexes were slightly hyperactive and the Oppenheim and Hoffmann signs could be elicited. There was papilledema of 1 D. on the right, and 2 D. on the left.

Ventriculograms indicated a tumor midway between the posterior portions of the bodies of the lateral ventricles. The roof of the 3rd ventricle was displaced downward, and the walls of the posteromedial portion of the lateral ventricles bulged outward. Calcification corresponding to the position of the pineal body also was visible.

Operation, Feb. 8, 1944. Craniotomy (E.H.C., assisted by Dr. J. L. Pool) via a right parieto-occipital approach. The falx cerebri was normal. Slight yellowish discoloration was noted near the splenium of the corpus callosum. The posterior portion of the corpus callosum was split in the midline for a distance of 3 or 4 cm. Beneath it was found material which at first glance resembled an organized hematoma, but further dissection showed it to be a well-encapsulated tumor. Extirpation was then undertaken. A 5 cm. cone of the right parietal lobe was removed from the post-Rolandic region to afford exposure of the right and superior aspects of the tumor; then, in order to gain access to the left side of the tumor, the lower two-thirds of the falx cerebri were divided, clipped and retracted. The corpus callosum was split further anteriorly. The tumor was found to be attached near the junction of the falx and incisura (Fig. 1A). At the attachment a vein, believed to be the right lesser vein of Galen, was found to traverse the tumor. The anterior pole, which extended fully 5 cm. anterior to the level of the incisura, was lifted from its bed, leaving the tela choroidea intact and exposed. On tipping the tumor, a thumb-sized posterior projection was seen to extend along the undersurface of the tentorium. Here arose a considerable portion of the tumor's blood supply.

The *postoperative course* was stormy, with prolonged unconsciousness and frequent fits involving the left side. On one occasion the wound was re-explored and old blood clots and necrotic tissue were removed. By Mar. 17, 1944, the patient was again alert and cooperative. The left hemiplegia had receded and sphincter control was being slowly regained. He was strong enough to walk, but was incoordinate on the left side. There was some reduction of hearing bilaterally. Improvement continued until April 26, 1944, when he showed unmistakable evidence of increasing intracranial pressure. A CSF fistula developed at the operative site. Death occurred on May 5, 1944, about 5 months after onset of symptoms and 3 months after operation.

Pathologic Examination. Microscopic. The tumor removed at operation revealed near the tip of its posterior extension, i.e., in the region where normally the pineal body is to be found, a number of arenaceous bodies, which were embedded in masses of large spheroidal cells separated by an anastomosing connective-tissue stroma bearing a variable number of small cells (Fig. 1B). The large (parenchymal) cells had fairly abundant loculated cytoplasm (Fig. 2A). The nuclei were relatively large and contained a delicate chromatin network and one or two conspicuous nucleoli, generally basophilic. The characteristics of the small cells varied with the field examined. They were most abundant in the vicinity of the erstwhile pineal body. Most of them resembled small lymphocytes, i.e., they had a densely chromatic spherical or ovoid nucleus which contained fine or coarse chromatin bodies, and cytoplasm which was scanty and usually homogeneous. In some regions there were cells that resembled large lymphocytes and sometimes plasma cells (Fig. 2A). In a few stromal areas there were, in addition, scattered small polygonal and elongated cells with vesicular nuclei and homogeneous eosinophilic cytoplasm. An occasional eosinophilic leukocyte was noted. There were a few abnormal mitotic figures. This portion of the tumor, regarded as a "pinealoma," was clearly demarcated from the remainder of the tumor (Fig. 1B).

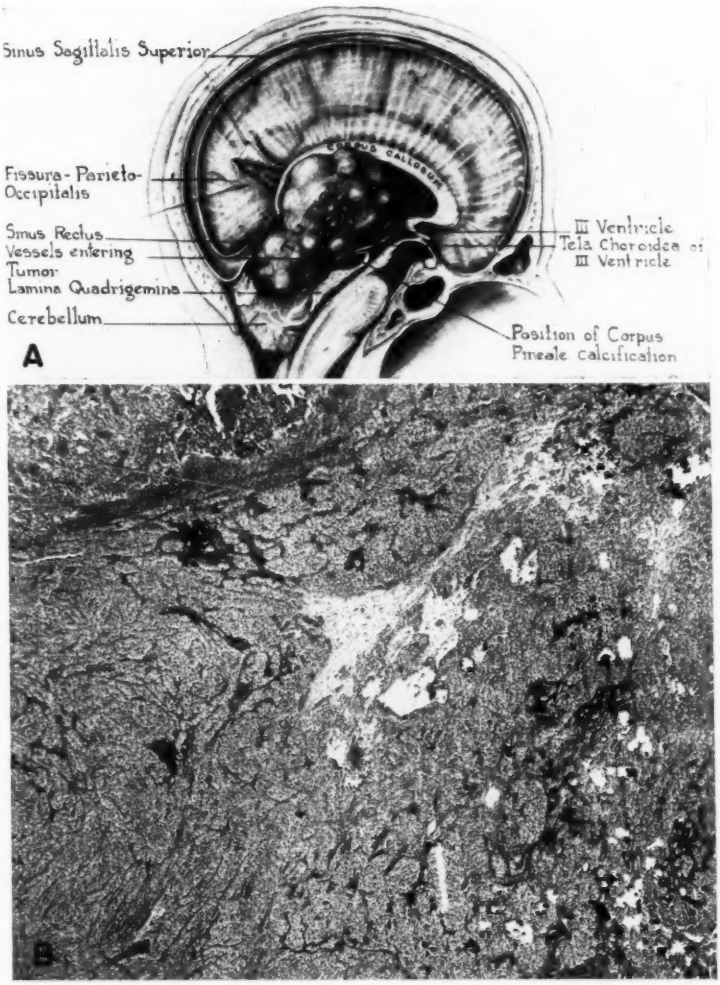

FIG. 1. *Case 1.* (A) Gross view, showing position of tumor. (B) Photomicrograph of tumor. Many arenaceous bodies (the small black structures) are embedded in a mosaic of large and small cells. This part of the tumor emerges abruptly with the carcinomatous part (upper left corner of field). A short distance beyond the field shown the tumor is a frank teratocarcinoma. Hematoxylin and eosin stain, ×18.

The great mass of the tumor had a varied structure. The predominant pattern was alveolar or papillo-alveolar, with a stromal core consisting of stellate or elongated cells overlaid by cuboidal or columnar epithelium (Fig. 2B); in many regions the epithelial cells were in wild disarray. Encountered here and there were foci of hyaline cartilage (Fig. 2B), large elongated cells in pseudo-rosette arrangement, groups of smooth muscle fibers, and structures resembling mucous glands of enteric type as well as serous glands and respiratory epithelium. No organoid structures were noted.

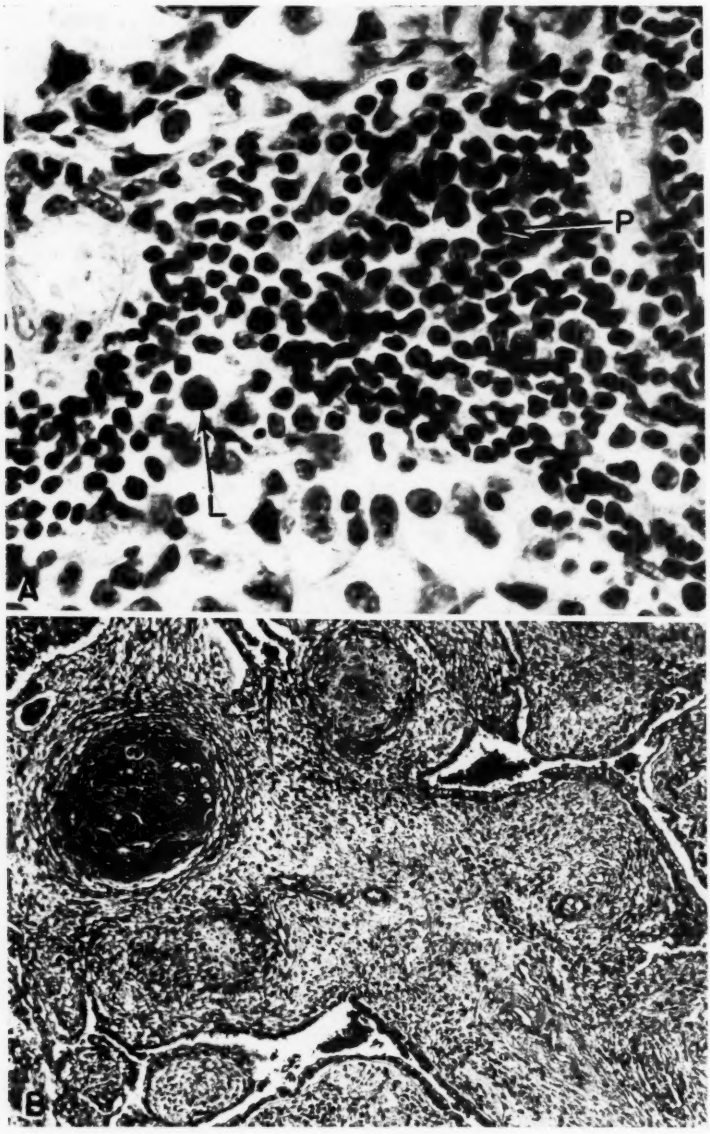

FIG. 2. *Case 1.* (A). Pinealomatous portion of tumor. The large cells have reticulated, slightly granular cytoplasm and relatively large vesicular nuclei possessing one or two prominent nucleoli. The small cells vary in appearance. Most of them are similar to small lymphocytes. Some resemble large lymphocytes (L), others plasma cells (P); still others are unidentified. ×700. (B) Teratocarcinomatous portion of tumor. The tissue is mesenchymatous and is arranged in alveoli, the surfaces of which are overlaid by cuboidal epithelium. Islands of cartilage are present. Hematoxylin and eosin stain, ×105.

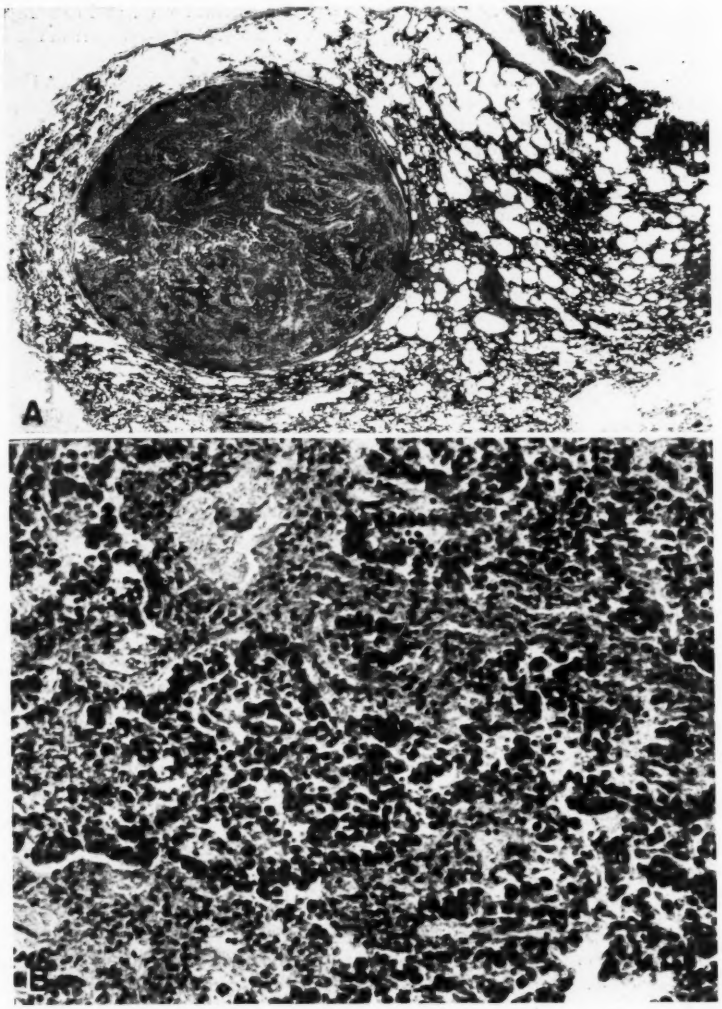

FIG. 3. *Case 1.* Pulmonary metastasis. (A) Gross view of one of the nodules. ×5. (B) Representative field of a tumor nodule. There is a mosaic of large and small cells in considerable disarray. A similar structure was seen in the portion of the intracranial tumor adjacent to the pinealoma (Fig. 1B), and within the straight sinus. Hematoxylin and eosin stain, ×200.

Autopsy. There was a massive recurrence of the tumor, with invasion of the centrum ovale of the right cerebral hemisphere above the lateral ventricle and extension into the corpus callosum as far forward as the level of the tip of the temporal horn. Moreover, the tumor had extended by way of the corpus callosum to involve the left hippocampal gyrus and the anterior portion of the left occipital lobe. The right lateral wall of the straight sinus was grossly invaded by the tumor; dissection revealed a solid mass occupying the lumen; it extended

rostrally into the vein of Galen and the right anterior cerebral vein, and caudally to the torcular. The lateral and 3rd ventricles were dilated. Histologically, the tumor in the straight sinus was regarded as a pineal carcinoma.

The lungs contained several nodules varying from 0.4 to 1.0 cm. in diameter. Some were deep in the pulmonary tissue, others near the pleura. Those deeply situated were spherical, while those on the surface were somewhat flattened externally. One of the larger nodules is shown in Fig. 3A. On section the nodules were found to be yellow-grey and crumbly. Each was surrounded by a narrow zone of subcrepitant dark red parenchyma. Microscopically the cells of the metastases closely resembled those of the intracranial "pinealoma," but architecturally (Fig. 3B) they were virtually identical with those in the portion of the tumor that had invaded the straight sinus. Abnormal mitotic figures were in profusion. The stroma was abundant and was permeated by small lymphoid cells; the larger lymphoid cells were relatively more numerous than in the primary tumor. The nodules were devoid of frankly teratomatous elements. The pulmonary growth was regarded as an alveolar carcinoma of pinealomatous derivation.

The remaining viscera showed little of consequence. Gross serial sections of the testes showed neither tumor nor scar, and microscopic examination also failed to reveal changes. The mediastinal, cervical, retroperitoneal and coccygeal regions were free from tumor, as were also the lymph nodes.

*Case 2.** A white male, aged 21, was admitted to hospital April 12, 1943 because of headache, diplopia, tinnitus and impairment of hearing, all of about 3 weeks' standing.

Examination. He was found to be mentally sluggish. There was marked papilledema with retinal hemorrhage, lateral rectus palsy and nerve deafness bilaterally, and slight weakness of the right side of the face. By April 16, 1943 his mental acuity was further depressed, and incoordination of the upper and lower extremities had developed. Ventriculography on April 19 revealed symmetric dilatation of the lateral and 3rd ventricles and evidence of a mass in the region of the pineal body which projected into the posterior portion of the 3rd ventricle.

Operation, April 23, 1943. Craniotomy† disclosed a large tumor in the region of the pineal body. It was partially removed. Histological diagnosis: "Pinealoma."

Course. On May 30, 1943, X-ray therapy was instituted, after which the patient steadily improved and became ambulant. Encephalography on July 5, 1943 disclosed a considerable reduction in the size of the tumor and of the ventricular system. Another course of X-ray therapy then was given.

The patient had improved considerably when, on Feb. 15, 1944, he began to complain of difficulty in walking, and shortly thereafter spastic paraplegia developed. The optic discs showed no abnormalities. CSF withdrawn on Feb. 8, 1944 was bloody and contained fragments of necrotic tissue in which neoplastic cells were identified. X-ray therapy was then applied to the vertebral column, with resulting improvement in the spastic paraplegia. However, the patient's general condition gradually deteriorated, death occurring Aug. 28, 1944, approximately 17 months after onset of symptoms.

Gross Pathology. In the pineal region there was a firm mass, $3 \times 2.5 \times 2$ cm. in size, which was adherent to adjacent falx cerebri and tentorium cerebelli. On section, its central portion was found to consist of cotton packing. Just in front of the mass was a discolored, calcified structure measuring $1.0 \times 0.8 \times 0.7$ cm. in size, which was regarded as a remnant of the pineal body. Within the 3rd ventricle and in the leptomeninges of the medulla oblongata, interpeduncular fossa, and base of the 3rd ventricle were tumor nodules varying in diameter up to 1.0 cm. Neither the spinal cord nor the hypophysis was removed at autopsy.

In the upper lobes of both lungs and in the lower lobe of the left lung were several well-circumscribed, firm, whitish nodules. Most of them were adjacent to the pulmonary surface. The largest, $3.0 \times 1.5 \times 0.6$ cm. in size, was situated in the left upper lobe close to the peri-

* Army Institute of Pathology Accession 95307.

† Not performed by one of us.

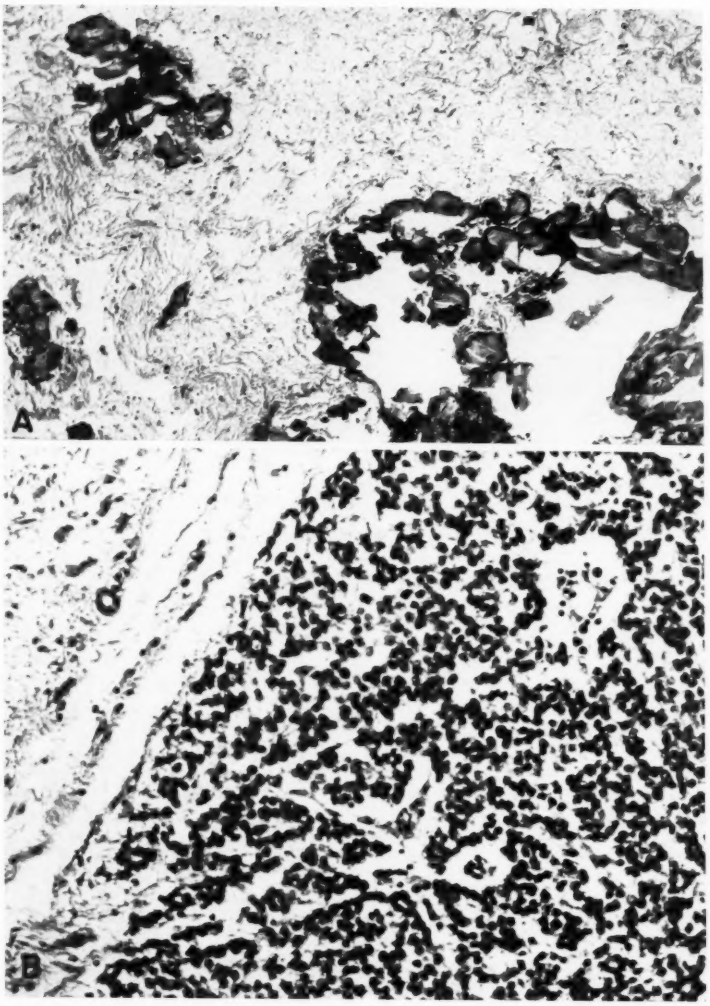

FIG. 4. *Case 2.* (A) Section through the pineal region. There are numerous arenaceous bodies in a loose fibrous matrix. ×75. (B) Section of a tumor nodule adjacent to dorsal portion of medulla oblongata. It consists almost exclusively of large (parenchymal) cells. Around some of the vessels a few small cells are present. Hematoxylin and eosin stain, ×175.

cardium. Several enlarged hilar lymph nodes were found. Section of them yielded homogeneous whitish masses surrounded by greyish, darkly pigmented parenchyma. Further examination revealed gross abnormalities only in the kidneys and urinary bladder: in the kidneys there were miliary greyish-yellow nodules in the cortex and reddish purulent streaks in the pyramids, and in the mucosa of the bladder a number of hemorrhagic and purulent foci. Some

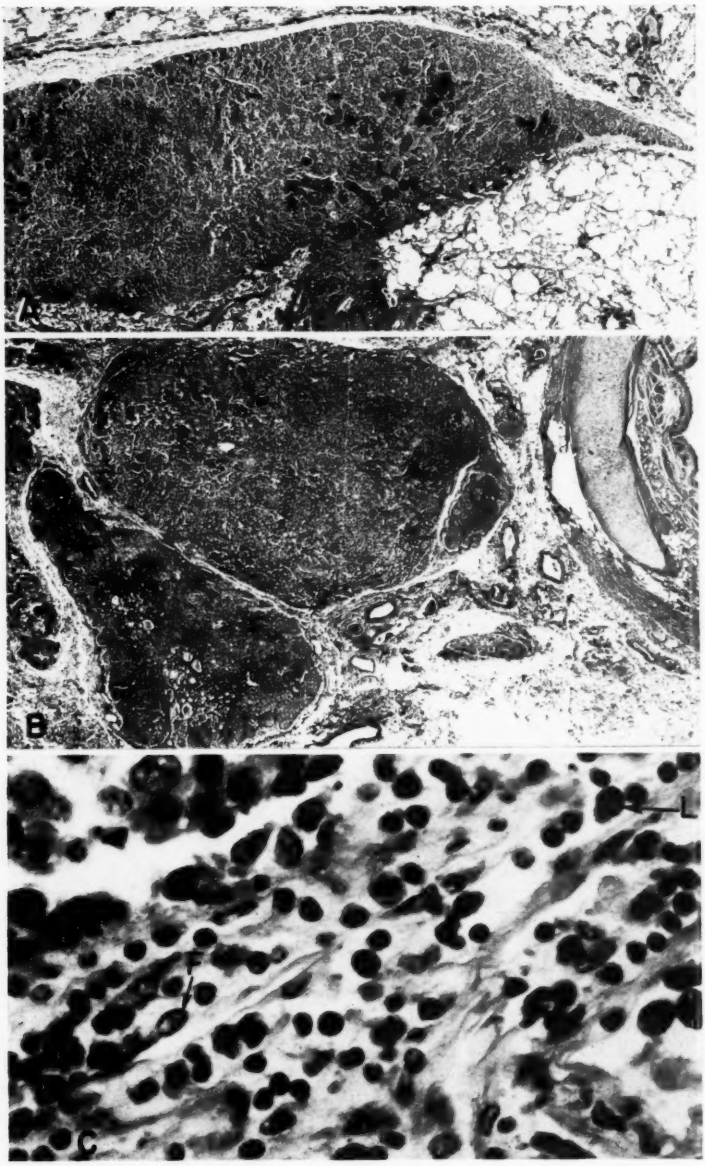

FIG. 5. *Case 2.* Metastases in lung and lymph nodes. (A) The major portion of the tumor occupies interlobular septa, but tumor cells are present also in alveolar sacs. ×10. (B) A hilar lymph node replaced by tumor, and one below it which is relatively intact. ×10. (C) Field from a pulmonary tumor. The large cells have abundant cytoplasm, and nuclei which are either deeply chromatic or vesicular. Most of these cells have inconspicuous nucleoli. The small cells vary in appearance. Many resemble small lymphocytes. A few simulate large lymphocytes (L) and fibroblasts (F). Hematoxylin and eosin stain, ×700.

of the mesenteric lymph nodes were enlarged, one being 2.0×0.8×0.3 cm. in size; section revealed "greyish-tan" parenchyma. None was removed for study.

Microscopic Examination. A study of sections through the retropineal region confirmed the presence of cotton fibers; they were surrounded by abundant fibrous tissue and many foreign-body giant cells. The small calcareous structure which at autopsy was thought to be a remnant of the pineal body was found to consist of dense fibrous tissue in which many arenaceous bodies were embedded (Fig. 4A). The original tumor, as well as the leptomeningeal and intraventricular nodules, was composed of masses of relatively large cells separated in places by connective-tissue stroma in which small cells were very sparse or non-existent (Fig. 4B). The nuclei of the large cells contained scattered clumps of chromatin, and the nucleoli were basophilic, usually of average size, occasionally large, and frequently not discernible; the cytoplasm was sparse. Pseudo-rosettes were frequent. There were numerous abnormal mitotic figures. In the lungs the tumor occupied alveolar sacs and interlobular septa (Fig. 5A). Tumor cells were present also in lymphatic channels of the lung. The affected lymph nodes were virtually replaced by tumor (Fig. 5B). Histologically the tumor nodules in the lungs and tracheobronchial lymph nodes were identical to the intracranial tumor as far as the large cells were concerned, even to the frequent lack of prominence of nucleoli, but differed in that many small cells were present in the stromal tissue (Fig. 5C). The small cells were predominantly lymphoid, but moderate numbers simulated large lymphocytes and plasma cells; also there were a few small polygonal cells with vesicular nuclei and well-defined eosinophilic cytoplasm.

Other findings were acute pyelonephritis and cystitis. In the testis there was suppression of spermatogenesis, only spermatogonia, Sertoli cells and a few spermatids being present. None of the other tissues showed abnormalities.

COMMENT

In both cases the evidence indicated that the intracranial tumor was primary and that the tumor nodules in the lungs were metastatic. In Case 1 the tumor was found to have invaded the straight sinus and grown within its lumen, whereas in Case 2 the site of entrance of the tumor into the blood stream was not ascertained. Moreover, in both instances the cytologic structure of the pulmonary tumors was sufficiently similar to that of the intracranial tumor to leave no doubt as to their source. Operative manipulation of the tumor in both cases may have been responsible for introduction of tumor fragments into the venous blood stream, resulting in the pulmonary tumors. Although these may not have been produced by natural mechanisms, they are none the less metastases. In the literature cited in the introductory paragraphs, operation had been performed in all except 4 cases and possibly 2 others in which the data on this point were indefinite.

The pineal body being such a small structure, it is not possible to determine the precise origin of the tumors, i.e., whether pineal or parapineal. However, in Case 1 it may be presumed that the tumor arose in the pineal body since arenaceous bodies were scattered in the tumor and since no normal pineal remnant was observed.

In Case 2 the arenaceous bodies were embedded in a mass of relatively acellular loose connective tissue, and there was no evidence of tumor in the immediate vicinity. It might be presumed that all tumor remaining in this region following operation was completely destroyed by X-rays.

The question arises as to the diagnosis of these tumors. In Case 1, in which the bulk of the tumor was regarded as a teratocarcinoma, a small part in the pineal region consisted of neoplastic tissue which had all the features of "pinealoma." Much of the metastatic tumor in the lungs was neither frankly pinealomatous nor teratocarcinomatous but represented an alveolar carcinoma composed of cells

having the appearance of anaplastic pinealomatous cells, such as were seen in a part of the intracranial tumor. In Case 2 the primary and metastatic tumors were pure "pinealomas," and differed only in that the metastatic tumor had a relatively much greater number of small cells than did the intracranial tumor.

There can be no doubt but that the small portion of the tumor in the pineal region in Case 1 and the entire tumor in Case 2 fall into the category of many of the "pinealomas" described by a number of authors.[2,13,14,22] Recently, however, the commonly accepted criteria of the "pinealoma" have been called into question. Dorothy Russell[20] has concluded that many so-called pinealomas are atypical teratomas, though true pinealomas also exist, a view concurred in by Friedman.[10] Since the "atypical teratomas" of the pineal body so closely resemble the seminomas of the testis, the two were regarded by these workers as analogous. Our Case 2 would fall into their category of atypical teratoma or seminoma, and Case 1 also, so far as its central part was concerned. Whether or not the viewpoint of Dorothy Russell and Friedman is justified will have to await further studies.

CONCLUSIONS

Two cases of tumor arising in the pineal body or its immediate vicinity and metastasizing to the lungs, are reported. In 1 of them a further extension to the hilar nodes occurred. In Case 1 a portion of the tumor which occupied the region of the pineal body had a pinealomatous structure, while the remainder was carcinomatous and teratocarcinomatous. In Case 2 the entire tumor had the features of a "pinealoma." In both tumors there were histologic structures which simulated those seen in the germinoma, or seminoma, of the testis.

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REMOVAL OF FOREIGN BODY (BULLET) FROM THE THIRD VENTRICLE*

James Greenwood, Jr., M.D.
Methodist Hospital, Houston, Texas

(Received for publication August 23, 1949)

Reports of movable foreign bodies in the ventricles are not numerous, and only one instance was found of the removal of a foreign body from the 3rd ventricle.[4] The clinical picture of intraventricular foreign bodies usually consists of repeated attacks of severe meningeal irritation or meningitis, ending in death if the offending object is not removed or, rarely, in recovery if the migration stops—the fragment becoming encysted somewhere in the wall of the ventricle.

Cushing[2] reported 30 cases of penetrating wounds of the ventricles with 8 recoveries; 16 of these were bullet wounds and all patients died. Regard[5] extracted a bullet from the lateral ventricle after it was made to drop posteriorly into the occipital horn. Small[6] reported a migratory bullet in the lateral ventricle which eventually came to rest in the substance of the occipital lobe and was not removed because there were no symptoms. Campbell, Howard, and Weary[1] reported a migratory buckshot in the lateral ventricle removed from the occipital region after a severe attack of meningeal irritation. Dandy[3] mentioned the removal of one bullet attached by a pedicle to the wall of the lateral ventricle and another freely movable which was removed through a ventriculoscope. Furlow, Bender, and Teuber[4] reported the removal from the 3rd ventricle of a large piece of shrapnel which had lodged there after migrating from the occipital region, presumably from the right posterior horn of the lateral ventricle. This is the only previously recorded case of a foreign body removed from the 3rd ventricle. It is similar in several features to the one which I shall present, particularly in regard to character change and rapid weight increase.

* Presented at a meeting of the Harvey Cushing Society, New Haven, Connecticut, June 2, 1949.

The following case report describes the removal of a bullet from the 3rd ventricle by an approach through the lamina terminalis, followed by complete recovery.

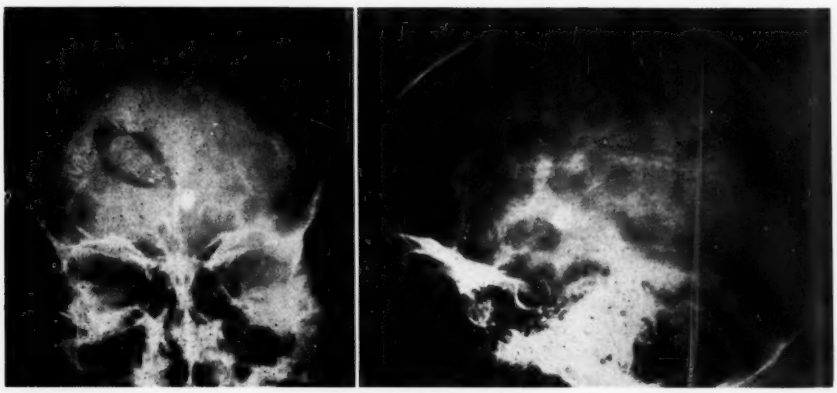
CASE REPORT

R.G.S., a 13-year-old boy, was admitted to the Methodist Hospital on Nov. 27, 1948, in a semiconscious state. On the day before he had received a bullet wound from a .22 caliber rifle fired at a distance of 75 to 100 yards, which entered his forehead just to the right of the midline. He was totally unconscious for about an hour, but then became semiconscious and able to talk, as well as recognize members of his family, although he was disoriented for time and place.

Examination. The vital signs were normal except for slight elevation in temperature. The right pupil was sluggish to light, and there was a partial right external strabismus. There was definite deviation of the tongue to the left, and weakness of the left arm and face, with almost complete paralysis of the left leg, but no significant reflex alteration except for bilateral Babinski sign and absent left cremasteric. X-rays showed the point of entrance barely to the right of the midline in the frontal region, a track of fragments, and the bullet near the midline on the right side, a little below the midpoint of the corpus callosum.

1st Operation. On Nov. 28, 1948 the wound was debrided through a small frontal opening. The dura was under considerable tension and a large clot was evacuated under it. The track of the bullet could be explored for a distance of 3 to 4 cm. before it was lost.

Course. He improved satisfactorily, became afebrile on the 7th day, and was able to use the left side of his body quite well. Diplopia persisted. Films taken on the 7th day showed a cylindrical metal object in the midline, measuring $1 \times \frac{1}{2}$ cm., slightly lower than in previous pictures (Figs. 1 and 2).



FIGS. 1 and 2. Anteroposterior and lateral views in usual positions. Bullet suspected of being in 3rd ventricle.

At this time he began to complain bitterly of pain in the neck, and there was considerable rigidity. X-rays showed a normal cervical spine. He began to run a septic temperature. On December 9, lateral x-rays taken in the brow-up and brow-down positions showed a midline movable bullet definitely in the 3rd ventricle, the position with the brow down being just above the optic chiasm, and with the brow up in the posterior recess or beginning of the aqueduct (Figs. 3, 4 and 5).

2nd Operation. On Dec. 14, 1948, 17 days after admission, he was taken to surgery again. The head was first placed in the brow-down position and then returned to a lateral position

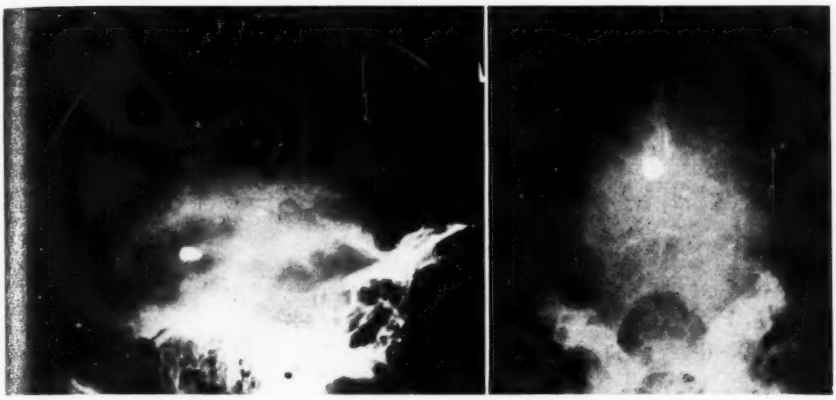


FIG. 3 (*left*). Lateral view, but with brow up. Bullet at entrance to aqueduct.
FIG. 4 (*right*). Anteroposterior view: bullet in aqueduct entrance.

with the brow still slightly down. Portable x-ray at the operating table showed that the bullet lay in the anterior portion of the 3rd ventricle.

A slightly longer incision was made in the right frontal region and another large segment of bone removed. The dura was well healed and under little tension. It was opened and the frontal lobe was gently separated from the floor of the anterior fossa, exposing the optic chiasm. A small opening was then made in the lamina terminalis above the chiasm, allowing escape of CSF. Exposure was not as good as had been anticipated, but the bullet could be palpated with a dull hook about $\frac{1}{2}$ inch behind the chiasm. With considerable teasing and a fervent prayer, the bullet was rolled over the chiasm. Cotton pledgets had been placed to prevent the escape of the bullet into the temporal fossa.

Course. For 2 days after surgery his temperature was between 102–103°, and he complained of considerable headache. After this, he improved rapidly and was discharged on the 10th postoperative day. There was still slight headache and diplopia. There was some left facial weakness, but no weakness of the extremities. He had been up and about for several days. X-rays showed the bullet now absent (Fig. 6).

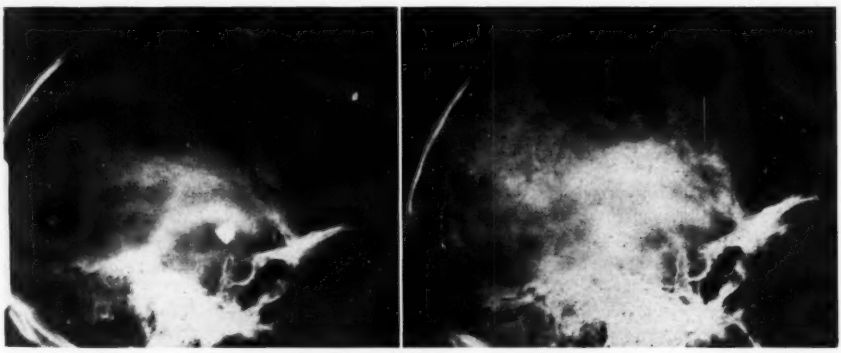


FIG. 5 (*left*). Lateral view. Brow down: bullet above chiasm.
FIG. 6 (*right*). Lateral view after removal of bullet through lamina terminalis. Fragments still present in frontal region.

Three weeks later, a grateful but rather alarming letter was received from his mother stating that the boy was fine except for the fact that he had an uncontrollable appetite and was drinking enormous amounts of water. She stated that he ate almost continuously during the day and got up several times during the night to raid the ice box. He returned for a checkup a week later and weighed 169 pounds, a gain of 36 pounds over his preoperative weight. Intake of fluids in 24 hours was 8250 cc. and output 8280 cc. He was quite obese—and even more striking was the tightness of the skin over the face, chest, abdomen, thighs, and buttocks.

FIG. 7. The patient 2 months after surgery.

He was referred to an internist for endocrinological studies. A glucose tolerance test, however, was entirely normal, and a routine urinalysis showed a specific gravity of 1.011. After a night of almost complete deprivation of water, the morning urine specimen showed a specific gravity of 1.020. On reduction of fluid intake alone, he lost 7½ pounds in a week, and it was concluded that this represented water loss, since his average food intake of 3500 calories had not been reduced. His caloric intake was then reduced to 2000 in 24 hours, and with phenobarbital, dexedrine sulfate, and considerable supervision he lost an additional 2½ pounds.

He has returned to school and is doing well in his studies. There were several episodes of bad temper at home, apparently cured by the proper paternal therapy. Behavior in school has been good (Fig. 7).

COMMENT

Whether the enormous appetite for food and fluids was part of a mental condition or a transient metabolic disorder cannot be determined, since it has cleared up and since the usual metabolic tests were normal 1 month after surgery. The parallel between this case and that of Furlow, Bender, and Teuber[4] is striking. Since no one will gain a wide experience in foreign bodies of the 3rd ventricle, thorough study of such a case is desirable. The type of surgical approach used, while advantageous from the standpoint of minimizing brain trauma, was not as easy as the transcortical or transcallosal approach used for cysts of the 3rd ventricle.

SUMMARY

1. A case of movable foreign body (bullet) in the 3rd ventricle and its extraction through the lamina terminalis has been presented.

2. Temporary postoperative mental or metabolic disturbances resulting in enormous appetite for food and fluids has been outlined.

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ENCASEMENT OF FOREIGN BODY (BULLET) IN THE THIRD VENTRICLE FOR 9 YEARS

CASE REPORT

MAX T. SCHNITKER M.D.*
Toledo, Ohio

(Received for publication July 14, 1949)

In rare instances of gunshot wounds of the head in which the bullet comes to lie in a cerebral ventricle (without infection) and without killing the patient, the question arises whether it is necessary or justified to remove the foreign body. Greenwood[1] points out that if the bullet migrates about the ventricular space, brain damage and reaction ensue, necessitating its removal. The following case report deals with a bullet that became encysted in the anterior portion of the 3rd ventricle. It produced so few symptoms that it was considered inadvisable to attempt its removal.

CASE REPORT

In January 1940, J.B., aged 11 years, got into a discussion with an older boy who had a gun and threatened him with it. The patient, in running away, turned to look back just as the gun was fired with the intention of shooting over his head, to scare him. The bullet (.22 calibre, short) struck the patient just to the right of the midline and above the glabella. The patient was very frightened, but did not lose consciousness. He was taken to a hospital.

FIG. 1. Bullet encysted in anterior third ventricle.

Examination. He was in mild shock and restless. Immediate neurological examination, done in reasonable detail, was entirely negative except for a questionable right lower facial weakness. Pupils were slightly dilated but reacted normally. Extra-ocular movements were full. There was slight bleeding from the bullet wound, but none from the nose. All reflexes, motor and sensory findings were normal.

Roentgenograms of the skull showed the bullet to lie exactly in the midline in the frontal plane. On lateral view it lodged about 2½ inches directly posterior to the supraorbital margin and in a vertical plane about midway in a line drawn from the bregma to the acoustic meatus. It was thought to be lodged in the corpus callosum and was "fixed" in position. Multiple minute bone fragments were embedded along the bullet channel above the olfactory groove. The frontal sinuses were undeveloped.

Treatment and Course. He was given intravenous fluids for shock and on the following day a debridement and closure of the wound of entry was carried out under local anesthesia. Lumbar puncture showed normal pressure, but the fluid was blood-tinged. Smear and culture of specimens of fluid were negative. The debrided tissue of the forehead showed pure culture of staphylococcus. Sulfanilamide, 1 gm. q 4 hours, was started on admission.

On the 3rd hospital day, patient complained of slight headache; there was rigidity of the

* 1031 Secor Hotel Bldg., Toledo 4, Ohio.

neck, and temperature was elevated to 101° (R). The wound was clean. Repeat lumbar puncture showed normal pressure; fluid was xanthochromic and sterile. On the 7th day, patient was more drowsy and showed a blood sulfa level of 8.5 mg. per cent. Skull x-rays were repeated and showed no change in the position of the bullet. On the 9th day he broke out in a generalized rash interpreted as due to the sulfa drug. Rbc was 3,350,000. The sulfa was discontinued and a transfusion of 250 cc. of whole blood administered. All symptoms cleared promptly. A detailed neurological examination done on the 17th day was entirely normal. He was discharged well on the 18th day.

Subsequent Course. Repeat x-rays (Mar. 1, 1940—5 weeks) disclosed that the bullet had moved downward and forward to a position directly above the posterior clinoid processes and posterior half of the sella turcica. It was concluded that the bullet was now lodged in the anterior portion of the 3rd ventricle. The latest film (Jan. 11, 1947) and fluoroscopy showed the bullet to have remained encysted in this position (Fig. 1).

By July 1941 (18 months), the patient began to have polydypsia and polyuria—about 3,500 cc. fluid intake/24 hours. By January 1947, this had slowly increased to 5,000 cc./24 hours, with urine sp. gr. 1.010. He was urged to use posterior pituitary substance by nasal insufflation, but this was refused as being unnecessary.

On last examination (May 7, 1949), 9½ years after the injury, he was entirely symptom free except for polydypsia and polyuria, which had increased to 10,000 cc./24 hours, with urine sp. gr. 1.001. He was 20 years of age, his height 64½ inches and weight 140 pounds. He is a sophomore at the University of Toledo and doing better than average work. General physical and neurological examinations are entirely normal. All secondary sexual characteristics are fully developed and he shaves daily. He voids every 2½-3 hours and nocturia is 1–2 times. He still insists this does not bother him sufficiently to warrant taking posterior pituitary substance.

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HYPEROSTOSING MENINGIOMA OF PTERION

CLAY MODEL AS AID IN SURGICAL EXCISION WITHOUT BONE FLAP*

Arthur Ecker, M.D.†
Syracuse, New York

(Received for publication July 14, 1949)

The difficulty in visualizing the hyperostosis affecting the four bony surfaces that results from pterional meningiomas *en plaque* is well known.[1] In such a case, invaluable assistance was offered by a prepared skull on which clay had been added to simulate the thickened bone. Furthermore, the orientation gained by using this model in the operating room permitted direct removal of the hyperostosis. Thus, even without a bone flap, there resulted sufficient exposure to permit both excision of the meningiomatous plaque and lateral decompression of the orbit. A report of this case is the basis of this paper.

CASE REPORT

History. The patient was a 48-year-old woman referred by Dr. Louis Alkoff. Her complaints were protrusion of the right eye ball and a mass in the right temporal fossa. Both conditions had been present for 8 months.

* Presented at meeting of the Harvey Cushing Society, New Haven, Connecticut, June 4, 1949.
† 608 East Genesee Street, Syracuse 2, New York.

FIG. 1. Lateral view of patient. (A) Pre-operative. (B) Postoperative.

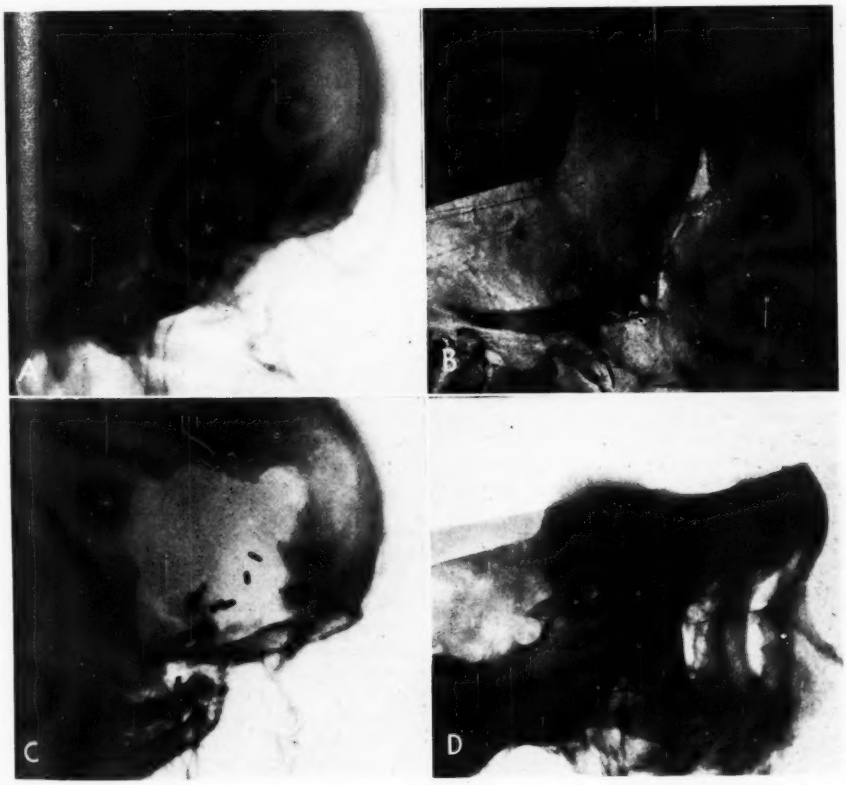

FIG. 2. Lateral views. (A) Pre-operative x-ray of patient's skull. (B) Photograph of model. (C) Post-operative x-ray of patient's skull. (D) X-ray of model.

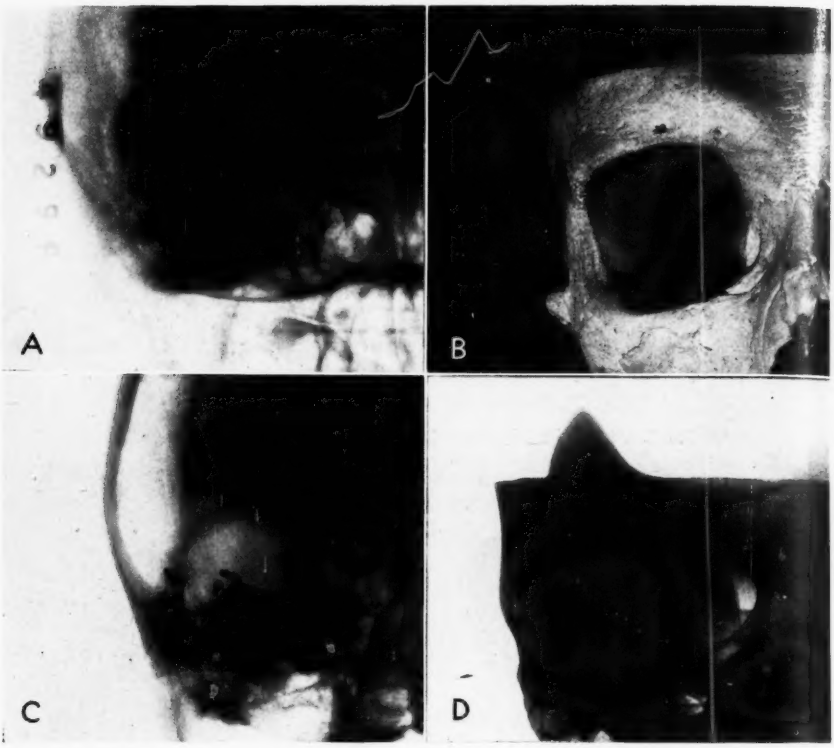

FIG. 3. Anteroposterior views. (A) Pre-operative x-ray of patient's skull. (B) Photograph of model. (C) Postoperative x-ray of patient's skull. (D) X-ray of model.

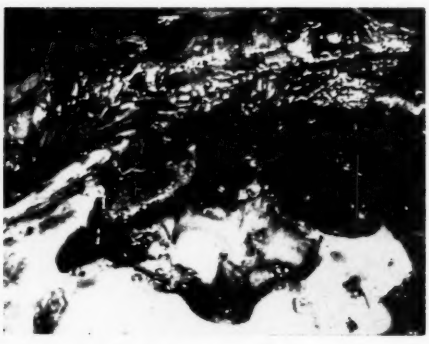

FIG. 4. View of operation. Hyperostosis in course of removal.

Physical Examination. There was a prominent bony mass, 5.5×3.5 cm. and raised about 1 cm., in the anterior part of the right temporal fossa (Fig. 1A) and moderate exophthalmos of the right eye (exophthalmometer readings: right eye—21 mm., left eye—12 mm.).

Roentgenograms of the skull revealed a hyperostosing lesion of the greater and lesser wings of the sphenoid bone on the right side (Figs. 2A and 3A). There was no evidence of involvement of the anterior clinoid process or the optic canal.

Preparation of Clay Model. Modeling clay was applied by Mr. Lee Brown Coye to half of the base of a prepared skull in

such a way as to simulate the hyperostosis (Figs. 2B and 3B). The model was checked fluoroscopically and roentgenographically (Figs. 2D and 3D) by Dr. Clayton Hale.

Surgical Technique. A partly oblique transcoronal incision was made entirely within the hair line of the scalp which then was reflected forward in two layers. The temporal muscles were retracted from the hyperostosis in the temporal fossa and that portion of the deep surface of the muscle that was invaded by tumor was removed. Excision of the hyperostosis in the temporal fossa, anterior fossa, middle fossa, lateral wall of the orbit, and part of the superior wall of the orbit was then carried out by means of a motor-driven burr and rongeurs

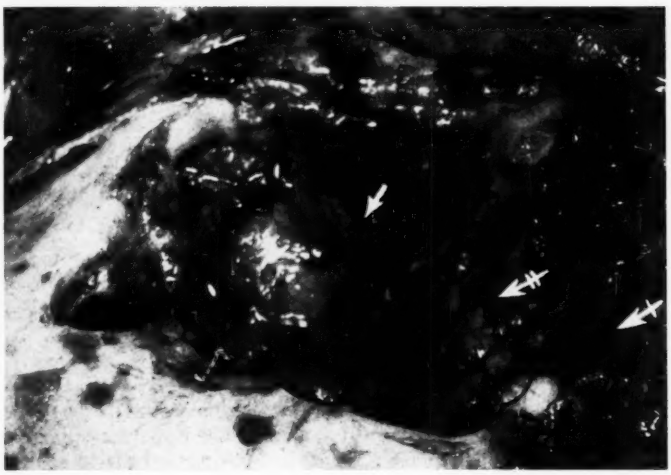

FIG. 5. View of operation. Orbital tissues (←) decompressed laterally. Pericranial graft (↔) sutured in position, making water-tight repair of dural defect. Temporal muscle (↔) after removal of meningiomatous infiltration from deep surface.

(Fig. 4). For this procedure, orientation was provided by the use of the clay model. The space obtained by removing the hyperostosed bone was adequate for excision of the meningiomatous plaque of dura in the middle fossa. This plaque measured 2.8 cm. by 4 cm. and varied from 3 to 5 mm. in thickness. The dural defect thus produced was repaired in water-tight fashion[3] by a pedicled graft fashioned from the deeper layer of the scalp, namely, pericranium and temporal fascia (Fig. 5). The graft was hinged at the supra-orbital ridge. Finally, at the site of removal of the hyperostosed bone, the lateral aspect of the orbit was exposed and the peri-orbita was incised in cruciate fashion, permitting decompression of the orbital contents (Fig. 5).

Microscopic study confirmed the diagnosis of meningioma and also meningiomatous invasion of the temporal muscle.

Postoperative Course. Convalescence was uneventful and the wound healed by primary intention (Figs. 1B, 2C, 3C). Exophthalmometer readings made 2 months after operation were: right eye—17 mm., left eye—12 mm. When seen 6 months after operation, the patient was feeling fine and was entirely free from symptoms. The pulsation at the site of the cranial defect was almost imperceptible.

DISCUSSION

Those meningiomas of the sphenoid ridge described as the pterional tumors *en plaque* or hyperostosing lesions of the ala magna were thoroughly described by

Cushing and Eisenhardt.[1] By the time the patient comes to surgery, the total excision of all involved dura and bone is so extensive as to be surgically impossible in most of these cases. It may therefore reasonably be asked, "Of what value is surgical intervention?" This question is especially appropriate because of the experience of Norman Dott[2] that these tumors are radio-sensitive. Apparently, surgery may be expected to do three things: (1) to relieve the exophthalmos, which is usually due to overgrowth of bone in the lateral wall and roof of the orbit; (2) to remove the deforming prominence of bone in the temporal fossa; (3) to remove that portion of dura that is involved by tumor, the presumed site of origin of the neoplasm.

For surgical management, Cushing employed a large osteoplastic flap involving most of the temporal fossa. He emphasized the excision of the portion of dura involved by tumor. Poppen and Horrax[4] made a frontal bone flap which resulted in exposure of the anterior fossa and permitted decompression of the roof of the orbit in order to produce recession of the proptosed eyeball.

In the case here reported, it was originally planned to combine the advantages of these two procedures by making a large fronto-temporal flap. However, the orientation gained from the clay model permitted a direct attack on the hyperostosis. After the thickened bone was removed, it was surprising to find enough room to permit the other two surgical objectives, namely, excision of the meningiomatous plaque and lateral decompression of the orbit. In similar future cases a shorter curved scalp incision should prove adequate.

SUMMARY

There is reported a case of hyperostosing meningiomatous plaque of the greater sphenoid wing. Modeling clay was used to simulate the hyperostosis on a prepared skull and proved invaluable for orientation in both pre-operative study and the actual course of the operation. The surgical approach consisted of direct excision of the hyperostosed portion of bone in the temporal fossa, anterior fossa, middle fossa, and posterolateral aspect of the orbit. Even without a bone flap, the space thus obtained was adequate for both excision of the meningiomatous plaque and for lateral decompression of the orbit. Furthermore, there was performed water-tight closure of the dural defect by means of a pedicled graft made of pericranium and temporal fascia.

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ASYMPTOMATIC TRANSFIXION OF SPINAL CORD BY A KNIFE BLADE

REPORT OF A CASE

RAFAEL CASTILLO, M.D., AND EDGAR A. KAHN, M.D.
Department of Surgery, Section of Neurosurgery, University Hospital, Ann Arbor, Michigan

(Received for publication July 21, 1949)

That a man can carry on his normal activity for a period of years with his spinal cord transfixed by a knife blade 2½ inches in length is almost unbelievable. The bizarre story of this colored laborer seems well worth recording.

CASE REPORT

H. S., a 43-year-old negro, entered the University Hospital Nov. 27, 1948 complaining of weakness of the left leg and increasing difficulty in the sexual sphere for 3 years. There had been gradual progression of these symptoms since their onset.

Past history revealed that he had been stabbed in the back 23 years previously in an altercation. He did not fall to the ground but walked to an automobile and was driven to a physician's office. There was considerable loss of blood but no pain, paralysis, or other disability. The next day he noted pain in the upper back which ran through to the chest but did not seem to radiate. The pain was constant and not shooting in character. It disappeared completely within a month. It is not known whether he had fever at any time but he did not have headache, stiff neck or photophobia.

Five weeks later the patient returned to light work. He noted then that the left leg was a little weak. The weakness had completely disappeared, however, by the time he had returned to heavy labor 3 months after the injury.

No difficulty was experienced for 13 years, when he noted on dancing that coordination was not as good with the left leg as it was with the right. His sexual life had remained perfectly normal.

Three years before admission he noted that orgasm was more difficult to obtain. He continued to have erections, however, with normal voluptuous sensation. In the last year orgasm was experienced about 3 times.

At about the time the patient first was conscious of his sexual difficulty he began to notice increasing stiffness and weakness of the left leg. This gradually progressed and on admission he walked with a limp but without the aid of crutches. No urinary difficulty was ever noticed. He claimed that he was able to tell the temperature of bath water with both feet.

Examination. Sensation: There was hypalgesia and hypesthesia to the level of D4, more apparent on the left. Deep pain sensation was diminished bilaterally but was more marked on the left. Vibratory sensation was absent in the left ankle and diminished in the right. Position sense was intact bilaterally. Combined skin writing was recognized poorly throughout but was probably impaired in the lower extremities. The cranial nerves were intact.

Motor: Patient walked without aid, but with a distinct limp with circumduction of the left leg. There was spastic monoplegia of the left lower extremity with atrophy and myokymic movements following exercises. Coordination showed some ataxia on left heel-to-knee test, probably a result of weakness. Romberg's sign was negative. The anal sphincter was intact. There were no signs of meningeal irritation.

Reflexes	*R*	*L*
BJ	++	++
TJ	++	++
RP	+	+
KJ	+++	++++
AJ	+++	++++
Babinski	Positive	Positive

	R	*L*
Abdominals	+	0

Clonus on the left, ankle and patellar.

Routine laboratory tests were not contributory and L. P. was not performed.

Roentgenograms of Spine. "Foreign body (broken knife blade), measuring approximately 3 inches in length, at level of D4 projecting through the neural canal to the posterosuperior aspect of D4 and lying just to the left of midline (Fig. 1). Localized destruction of the inferior portion of the lamina of D4 and the superior portion of the lamina of D5 around this foreign body."

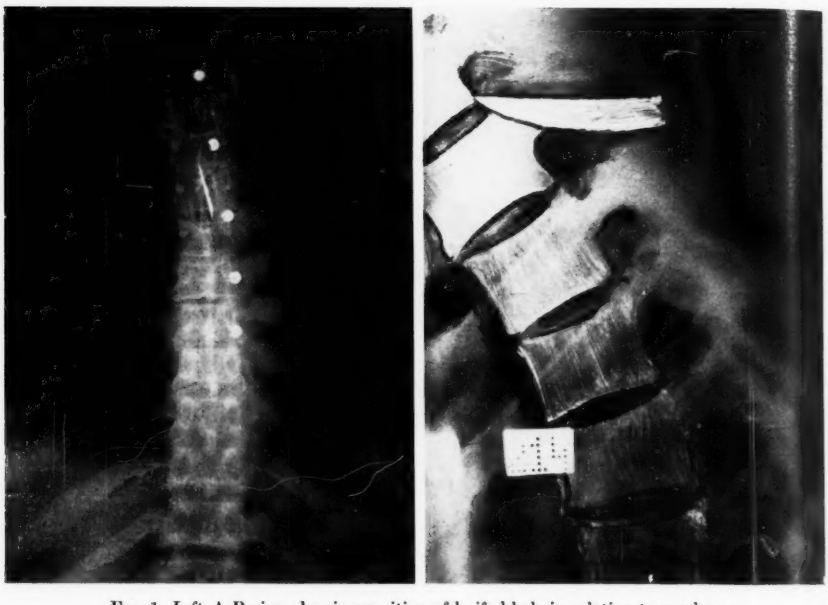

FIG. 1. *Left,* A-P view showing position of knife blade in relation to markers. *Right,* Lateral view (retouched).

Operation. By means of markers placed upon the skin and checked by x-ray, the exact position of the knife blade was determined and marked upon the skin. For the first time now the small, fine scar of the wound of entrance was seen. A vertical incision was made centering over this area. The 4th and 5th dorsal laminae and spinous processes were exposed subperiosteally. At a point in the fascia just to the left of the 4th spinous process, a discoloration was seen. It was realized that the knife blade lay beneath this point. By sharp dissection the upper part of the knife blade was exposed. It was adherent to the 4th dorsal spinous process. It was grasped with forceps and extracted with a moderate amount of force. There was an immediate escape of dirty, rusty fluid followed by a flow of CSF. It was seen that a meningocele sac had formed around the knife blade. The 3rd lamina was now removed, exposing normal dura at the upper part of the incision. A laminectomy was then performed down through D5. There was a mass of scar tissue adherent to the dura in the midline where the knife blade had been extracted. The dura was now opened below without opening the arachnoid. The scarred dura was excised, leaving the arachnoid intact. The dural incision was then carried in the midline up almost to the 2nd lamina. A mass of grayish-black scar tissue lay in the cord substance in the midline. It seemed fairly well encapsulated and was part of the sinus tract. It was firm in consistency. The cord substance around it, however, seemed softened and was yellow. This mass lay exactly like a small midline intramedullary tumor, and measured 0.8 by 2.0 cm. It was completely removed (Fig. 2). It could now be established that the knife

had entered the spinal cord in the midline and had completely pierced it and had reached the anterior part of the vertebral canal where it was imbedded. There was an actual defect in the spinal cord anteriorly about 4 mm. in length, exactly in the midline, and a small instrument could be passed through this defect down to the anterior dura. It looked as if the posterior columns had been completely destroyed. The lateral columns, however, appeared to be in fair condition. The attachment of one dentate ligament was divided so that the cord could be thoroughly explored. There was no question but that the knife had passed completely through the center of the spinal cord. After careful hemostasis the upper and lower parts of the dural incision were closed. There was a small defect left, however, which was covered with two separate layers of gel foam. The muscles were now brought together with silk and the skin closed in the usual manner.

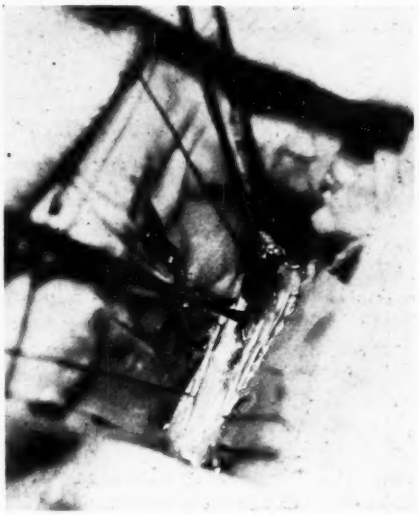

FIG. 2. Operative findings. The dark mass is the scar tissue which has been freed from the dura and dissected from its intramedullary bed.

Pathological Report (Dr. Robert Lam). "Sections stained by hematoxylin and eosin and Heidenhain's connective-tissue method demonstrate an extremely dense cicatrix containing iron rust and hemosiderin, and surrounded by a thin layer of compressed and degenerated spinal cord. Lining the scar, the rust appears as irregular, highly refractile, sharp-edged yellow or green particles that are either crystalline or granular in form. Large oval or elongated aggregates of iron rust are present in the connective tissue, which has been laid down in a more or less concentric lamellar manner. There is a diffuse staining of the inner edges of the scar which gives a positive reaction to the Prussian blue test. Phagocytized and nonphagocytized hemosiderin is scattered among the masses of iron rust. There is also abundant hemosiderin in the external portions of the scar and between the degenerated myelin fibers of the adjacent spinal cord. Marked iron reaction in the vessel walls is present in areas of vascular proliferation in the connective tissue. A moderate degree of lymphocytic perivascular infiltration is encountered in the blood vessels in the outer margins of the cicatrix. There is no evidence of any glial reaction."

Postoperative Course. The patient did very well and was able to move both legs. There was marked spasticity of the lower extremities. There was posterior column involvement, vibratory, position sense, deep pain and skin writing being absent. Superficial touch was intact. Temperature and pain sense were absent to D4. He had retention of urine and was maintained on intermittent bladder irrigation drainage for 6 days, after which he had normal bladder function and good sphincteric control. On discharge (20 days after operation) he still had considerable weakness of both lower extremities but was able to walk with some assistance.

The patient returned for check-up 7 months after operation. He could now walk by himself using only one cane. There was spasticity in both legs, more marked on the left, but Babinski sign was bilaterally absent. Vibratory sense was present over both lower extremities and position sense was normal. The level of analgesia remained at D4. There was thermodysesthesia over the same area. He had had erections without orgasm. He has had good sphincter control.

Addendum. The patient was seen again 14 months postoperatively. He no longer needs

a cane to walk in the house. He has walked 7 hours a day outdoors in an attempt to gain strength. He has normal sphincter control. There is no improvement in sexual function. Sensory findings are unchanged. He has nevertheless made considerable improvement.

SUMMARY

A case has been presented in which the upper dorsal spinal cord was transfixed by a knife blade for 19 years before symptoms finally appeared. With the intense reaction in the spinal cord found at operation this patient's long period of normal activity is indeed surprising.

CONTINUOUS REGIONAL SYMPATHETIC BLOCK; DIRECT CATHETER TECHNIQUE

FRANKLIN ROBINSON, M.D.,* AND GERALD F. WHALEN, M.D.
Neurological Division, St. Vincent's Hospital, New York, N. Y.

(Received for publication July 20, 1949)

Procaine block of the sympathetic nervous system is an integral part of the evaluation and treatment of certain neurovascular disorders of the extremities. Continuous regional physiological suppression of the sympathetic pathways during a controlled period would be of further aid in the management of thrombophlebitis, peripheral vascular occlusion with vasospasm, causalgia, and the sympathetic reflex dystrophies, as well as in the selection of cases favorable for operation.

The advantages of such a technique would be (1) the elimination of frequently repeated injections and (2) the more accurate appraisal of effect when the patient's suggestibility associated with the routine procedure has been circumvented.

The caudal route[1,2,3] has been suggested and used for continuous lumbar sympathetic block. A direct catheter method of producing continuous paravertebral sympathetic block at any indicated level is the subject of this report.

TECHNIQUE

The method requires the use of a special thin-walled blunt needle equipped with a sharp pointed inner obturator and a segment of small-bore vinylite plastic tubing which can be passed readily through the needle (Fig. 1). A No. 18 needle, 9 cm. in length, is suitable. The

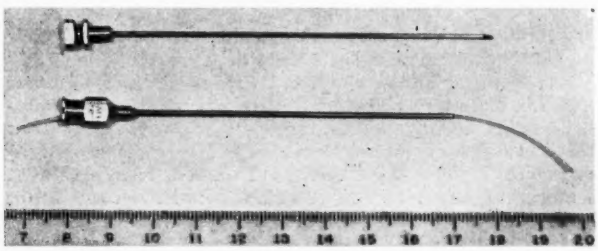

FIG. 1. Obturator and needle with plastic tube inserted.

* 333 Cedar St., New Haven 11, Conn.

shaft and hub of the needle are soldered externally rather than riveted so as to have an uncompromised large bore in order to facilitate free passage of the plastic tubing. The vinylite tubing is 12 to 14 cm. in length and will accommodate the insertion of a No. 23 needle.

Procedure. Paravertebral block is performed in the conventional manner with sterile precautions. For lumbar sympathetic block, the level of the 2nd lumbar ganglion usually is selected. In stellate ganglion block the anterior or posterior approach may be used, the latter being favored by the authors. The needle is inserted in the usual manner to the paravertebral region at the desired site. The obturator is removed and, after preliminary aspiration as a precautionary measure, 10 cc. of 1 per cent procaine hydrochloride are injected slowly. The solution is allowed to diffuse through the tissues for approximately 10 minutes. Success of the block is judged at the end of this period according to the usual criteria. When satisfactory block is achieved, the vinylite tube is passed through the needle to its tip. The needle is removed by withdrawing it slowly with a twisting movement while the tube is kept in its original position with gentle pressure. The tube is fixed to the skin with a fine silk percutaneous suture passed through the procaine skin wheal (Fig. 2). The exposed tubing and injection site are protected with a sterile dressing. It may be necessary to inject two levels in the lumbar region, L2 and L3, in order to obtain full effect, and, in these cases, twin catheters are used. At intervals of 2 to 6 hours as determined by the clinical response, the dressing is removed and 10 cc. of procaine are instilled into the catheter by syringe with a No. 23 needle. The patient's activity in bed need not be limited.

FIG. 2. Plastic tube in position for continuous stellate block.

Sterilization of Equipment. The vinylite plastic tubing (boilable) and needle are sterilized by autoclaving and are included in the regular paravertebral block set.

Complications. It has been found important to anchor the tube to the skin with a suture in order to prevent its movement. In 2 cases where fixation with adhesive tape was relied upon, the tube was prematurely extruded in one instance and drawn into the paravertebral musculature necessitating operative removal in the other. Suture fixation of the tube has proved uniformly effective in all cases. Occlusion of the tube by kinking or by clotted blood has not been a problem and no special measures have been required to maintain its patency. Local infection has not been encountered. Continuous sympathetic block has been maintained for 72 hours with the indwelling catheter. If therapy is desired for a longer period, it is recommended that the tubing be changed and an adjacent segmental level be used.

COMMENT

This procedure has been successfully employed to provide continuous stellate ganglion block in cerebral thrombosis and in post-traumatic sympathetic reflex dystrophy of the upper extremity, and lumbar block in the treatment of thrombophlebitis, varicose ulceration, and intractable pain in the lower extremities. It is believed that the method affords an effective means of attaining continuous regional sympathetic block for both diagnostic and therapeutic purposes.

The authors are indebted to the Research Division, Becton, Dickinson and Company, for their generosity in providing the necessary equipment.

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THE PROBLEM OF THE GLIOBLASTOMAS*

ERNEST SACHS, M.D.†
New Haven, Connecticut

(Received for publication January 13, 1950)

THE YEARS 1918 and 1926 are two important dates in the development of neurological surgery. Therefore, in studying the problem of the glioblastomas, we must first consider what conditions we faced prior to 1918 and then again what influenced this problem after 1926.

In 1918 ventriculography was discovered. Prior to that date, those few of us who were attempting to do neurological surgery were occupied primarily with detailed neurological studies to enable us to arrive at a diagnosis of brain tumor, and then we carried these examinations still further trying to localize the lesion. When we had finally exhausted all our methods of study, which at that time consisted solely of the history and neurological examination, we had the further problem, no mean one at that time, of convincing our neurological or medical colleagues that an operation should be undertaken; and then, finally, we had the problem of the operation itself. It is difficult for neurosurgeons today to realize what we were up against. For example, the first dozen patients with brain tumor I saw in 1911 were all blind. I wondered whether I would ever see one before he was blind. In those days, we never dared to make a positive diagnosis of tumor unless the patient had a full-blown choked disc. The only exception to this rule was if we were fortunate enough to have a patient with typical Jacksonian convulsions. I do not remember just when I operated upon the first patient with tumor who had no choked disc, but it was after 1918. Though we made most careful neurological examinations, many of the symptoms and signs elicited we were unable to interpret correctly. For example, paresis of one sixth nerve caused us no end of difficulty and mental juggling trying to fit it into a clinical picture. We did not appreciate that bilateral sixth nerve paralysis was a very different matter. While unilateral sixth nerve paresis was a general sign of increased intracranial pressure due, as Cushing pointed out in 1911, to pressure on the nerve by a branch of the basilar artery, bilateral sixth nerve paralysis was due to a pontine lesion where the two sixth nerve nuclei lie close together. It took some of us some time to realize what a great difference existed between paresis and paralysis of these nerves and how seriously this might affect the diagnosis and prognosis in a given case.

The significance of visual fields was by no means clear, and the sign of a partial or complete homonymous hemianopsia we didn't learn to interpret until after 1911, when Adolph Meyer and Archambault described the visual

* Presented at the annual meeting of the Philadelphia Neurological Society, Philadelphia, Pennsylvania, January 6, 1950.

† 333 Cedar Street, New Haven 11, Connecticut.

pathway traversing the temporal lobe. It was only then that the importance of very careful study of the visual fields was appreciated. Even nystagmus and ataxia caused us difficulty, and I recall vividly exploring a patient's cerebellum twice because of these signs, and only at a third attempt, because of some other signs that had developed, did I finally remove a large frontal meningioma. Frontal and cerebellar lesions were not infrequently confused and v. Monakow advanced the theory of diaschisis to explain such occurrences. These are but a few of the diagnostic pitfalls we encountered.

Then came the operative procedure. Though this is still a formidable affair, in those days it was far more so, for we lacked many of the aids we have today. Blood transfusion was just beginning to be used, but it was unusual to have more than one pint ready, and of course that was long before the days of blood banks. Matching of blood was not well developed and citrated blood had not been heard of. So we had to use either direct transfusion by the Crile method or Kimpton tubes, waxed cylinders in which the blood was collected and then introduced into the patient. The control of hemorrhage was an ever-present problem. Wax to control bleeding from the bone and silver clips to control bleeding from the brain vessels we did have. Bits of muscle were also used as a hemostatic, taken either from the patient himself or from another being operated upon at the time, or by using pigeon's breast, as was practised by de Martel. The value of giving the patients fluid during the operation to counteract the fluid loss was not known or appreciated, and the control of increased pressure by tapping the ventricles had not become a routine procedure. In fact, I recall a meeting held here in Philadelphia in 1922 at which there was a lengthy discussion on the wisdom of doing a ventricular puncture in the course of an operation, and there were those who strongly opposed it.

Then it happened innumerable times that the patient was operated upon but the tumor was not found. No neurosurgeon today can fully appreciate the chagrin and mortification of being obliged to acknowledge *that*—time and time again.

When we did remove the tumor, the pathologist told us it was a glioma.

In 1918 Dandy made his epoch-making discovery of ventriculography. This was not immediately accepted, but as soon as it was, it transformed the matter of localization completely. It soon became almost a disgrace not to locate a tumor at operation, and negative explorations were far less frequent. After ventriculography came into general use, the incidence of locating a tumor jumped from about 55 per cent to over 97 per cent, but there still were tumors that we did not expose because we did not have facilities to incise the cortex freely, and therefore hesitated to incise it with impunity as we do today.

The pathologist still reported gliomas, but from this time on we removed tumors much more frequently and came to realize that though the pathologist reported all the tumors as gliomas there were different types. Some were cystic, some were well encapsulated, some contained calcium and

threw a shadow on the x-ray plate. The patients with the cystic, the calcified, and the well encapsulated tumors had a much better prognosis, and, even if the tumor had not been completely removed, had a longer survival period. We found that by combining a decompression with the operative removal, we were able to make many of these patients comfortable and get a certain number back to work, and we continued to follow this policy from 1918 to 1926. Then in 1926, Cushing and Bailey published their valuable monograph in which, by making use of the special stains of Cajal and Hortega, they were able to differentiate and classify the various tumors of the glioma group. They found that in the cystic tumors certain cells predominated and these they called astrocytes and the tumors astrocytomas. Another type of cell was found in the calcified tumors which they called oligodendrogliomas. In still another type that proved to be radio-sensitive, they identified medulloblasts and called the tumor medulloblastoma. Still another tumor was recognized that was rapidly growing and had a very short history, and this was called a glioblastoma.

These glioblastomas were, and still constitute, the most serious problem for the neurological surgeon. There are several fundamental principles that are involved in dealing with them.

First. Are we able to determine prior to operation the pathology of a tumor? The history of a glioblastoma is not characteristic but there are certain features that strongly suggest the diagnosis. If an adult patient has symptoms of increased intracranial pressure rapidly, in a month or two, and if the symptoms and signs indicate that the tumor is in the cerebrum, he is very likely to have a glioblastoma. I say adult because, though this type of tumor does occur in children, it is far less common than in adults, and I emphasize cerebrum as glioblastomas occur far more commonly above the tentorium than below it. In my own series there were 233 cerebral glioblastomas and only 7 cerebellar. Still, on the history alone, we would not be justified in making such a diagnosis. But it is well to remember that glioblastomas do constitute about 45 per cent of all brain tumors. Recently, some roentgenologists claimed to be able to identify a glioblastoma in an angiogram by the vascular pattern.

Second. What is the best method of handling a brain tumor today? There is no question that all benign tumors and well encapsulated tumors should be removed surgically. Even if the tumor is extraordinarily large or if it is located in a somewhat inaccessible place like the lateral ventricle, the third ventricle or the fourth, it should be removed. Some meningiomas undergo malignant degeneration and recur. Some oligodendrogliomas cannot be completely removed and sooner or later begin to grow again. Medulloblastomas, which are very radio-sensitive, may be completely removed at times but more often are incompletely removed and then are controlled with deep x-ray therapy for a number of years before they cause renewed trouble. But all neurosurgeons are agreed that all these tumors should be operated upon and an attempt made to remove them. *I* have always taken the same

attitude in regard to glioblastomas. Though I have rarely if ever permanently cured a patient with glioblastoma, I firmly believe these patients should be given relief, and relief they most certainly can get, sometimes for a number of years.

I have seen patients who, completely incapacitated temporarily, were entirely restored to a normal state so that they were able to carry on their work for a year or more. I recall a physician who had hemiparesis and was partially aphasic who was restored so that he was able to carry on his practice for a full year. This does not happen in many cases but sufficiently often, I believe, to justify the procedure. Then, too, most of the patients can be kept from becoming blind and be relieved of the excruciating headaches from which one with brain tumor suffers if operation is not performed.

By means of electrocoagulation and suction and the use of hemostatics like fibrin foam or gelfoam, more and more radical removals can be undertaken. But up to the present time, a permanent cure of a glioblastoma has rarely if ever been obtained. We therefore are faced with the third important question, and this involves an ethical as well as a purely medical problem.

Third. If we can determine that we are dealing with a glioblastoma that cannot be cured, should we do anything for such a patient? A group of younger neurosurgeons has in the past 5 or 10 years taken the attitude that if they can positively determine they are dealing with a glioblastoma, there is no use in operating. Some of them arrive at the diagnosis by a needle biopsy and others by an angiogram. To my way of thinking, this point of view cannot be too severely condemned. First of all, these men evidently do not believe that giving patients temporary relief is worth while, and therein are neglecting one of the important functions of a physician. Of course we would all prefer to operate only on those patients whom we can cure, and it is distressing to see patients return with a renewal of their symptoms, but does that give us the right to refuse them temporary help? Then too, neither a pathological diagnosis nor angiography is absolutely infallible. In the same way, I am entirely opposed to giving a patient deep x-ray therapy without first having a pathological diagnosis. I recall a lovely girl some years ago who had symptoms of a cerebellar tumor. The surgeon was so certain she had a medulloblastoma that he gave her deep therapy. She improved temporarily, and this was construed as confirmation of the diagnosis; therefore when her symptoms recurred, she was again given a course of x-ray therapy, but this had no effect. I saw her when she had marked impairment of vision, advised operation and removed a cholesteatoma. Her vision was saved and she has been perfectly well since then. This and similar experiences have made me feel very strongly that no patient should ever receive x-ray therapy until a pathological diagnosis has been made.

Lastly, this point of view, of not operating in these cases, is that of the defeatist, an attitude that I have never been able to accept. Had medicine followed this line of reasoning, progress would have been markedly hindered. Certainly many of you recall the days where there were no cures of carcinoma

of the stomach or the lung. Seventy-five years ago, Billroth wrote with distress of the high mortality he had in doing laparotomies for ovarian tumors but that did not deter him from continuing to try to remove them. Countless other examples could be cited in medicine and surgery where a defeatist attitude would have delayed progress.

The sound method of dealing with the glioblastomas seems to me therefore, until some more effective method of treatment is discovered, to continue to attempt to remove these tumors surgically, always aiming to be radical enough to try to obtain a cure. With this idea in mind, we must make more strenuous efforts and make use of every new device that is offered.

The greatest stumbling block in the cure of glioblastomas has been the difficulty of finding the limits of an infiltrating tumor. The electrosurgical unit and suction have been of inestimable help but they are not enough. A very promising aid lies in the recent discovery by Selverstone and Sweet that a tumor can be impregnated with radio-active phosphorus and then its limits be outlined by the use of a specially constructed Geiger counter.

Certainly we cannot hope to solve the glioblastoma problem by throwing up our hands and saying there is nothing we can do. On the contrary, the solution lies in our constantly pressing on, making more and more strenuous efforts to remove these tumors, and not allowing ourselves to be deterred by any obstacles that lie in our path.

EPIDERMOIDS: CLINICAL EVALUATION AND SURGICAL RESULTS

FRANCIS C. GRANT, M.D., AND GEORGE M. AUSTIN, M.D.*
Department of Neurosurgery, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania

(Received for publication August 7, 1949)

THE "EPIDERMOIDS" or "cholesteatomas" often present opportunities for complete removal and cure. This is due to their frequent epidural position, avascular character, and extremely slow development. Our paper deals with the surgical treatment and results of the group of 22 "epidermoids" verified on the Neurosurgical Service of the Hospital of the University of Pennsylvania and the Graduate Hospital of the University of Pennsylvania since 1930. It is not intended at this time to go into the slightly controversial etiology of these tumors, as they are now generally accepted to be the product of epithelial cell rests. Discussion has arisen concerning the better name for these tumors. At present, the term "cholesteatoma" is synonymous with "pearly tumor" and "epidermoid," the latter being the term of choice.[5,15,18] A difference of opinion also arises as to whether the cholesterin-containing masses, arising in the petrous portion of the temporal bone, should be classified as true tumors of the cholesteatoma group. The majority of pathologists believe these cholesteatomatous masses, often associated with chronic otitis media, to be unrelated to true epidermoid tumors. They are merely the result of some chronic inflammatory desquamative process.[3,18] These are best designated by the term "cholesteatoses."[2] The fact that both "epidermoids" and "dermoids" contain cholesterin often serves to confuse their differential diagnosis. The differential point is that the dermoids contain hair or other dermal elements due to their mode of origin. Critchley and Ferguson[5] pointed out that both occur as the result of a fetal inclusion of epidermal cells which, depending on the depth of the layer or according to their embryonic age, produce an epidermoid or dermoid type of tumor.

A complete review of the literature is not intended at this time, and one is referred to the more exhaustive works of Mahoney,[12] King,[10] and Rand and Reeves.[18] However, a brief historical outline is included to reveal the development of terminology and theory of origin of these tumors.

1807—Pinson,[17] an artist in the School of Medicine of Paris, first described an epidermoid tumor from Dupuytren's service at the Hôtel Dieu.

1829—Cruveilhier,[6] in his *Anatomie Pathologique*, fully described the tumor of Pinson and also a parapituitary tumor of the same type. Because of their highly refractile and nodular surface, he gave them the name, *tumeur perlée.*

* Francis Clark Fellow in Neurosurgery.

1838—Müller[14] described 3 similar tumors and, because of their content of cholesterol crystals, called them "cholesteatomas." This has remained the term of popular usage in spite of the fact that it is neither complete nor accurate. Horrax[9] pointed out that the dermoids frequently contain cholesterol crystals and still possess the pearly sheen of the closely related epidermoids.

1854—Remak[19] proposed the theory that these tumors arise from epithelial cellular "rests."

1877—Perls[16] suggested the possibility of an endothelial origin of the "cholesteatoma."

1897—Bostroem[4] introduced the terms, "pial epidermoids" and "dural dermoids."

1920—Bailey[1] described the first 2 cases of successful surgical removal of intradural epidermoids.

1928—Martin, Dechaume, and Puig[13] discussed the 2 outstanding theories of development: namely, that they result either from a fetal inclusion of epithelial cells, or from metaplasia of endothelial cells of the meninges.

1928—Critchley and Ferguson[5] refuted the endothelial theory of origin. They reiterated the generally accepted theory that the epidermoid or dermoid tumors were both the result of fetal inclusion of epidermal cells, depending on the depth of the layer or according to the embryonic age.

1936—Mahoney[12] reported on 142 epidermoid tumors which he had collected from the literature.

1943—Rand and Reeves[18] stated that less than 200 epidermoids had been reported, and at the same time added 19 cases of their own. However, 4 of these were accidental findings at necropsy.

INCIDENCE

The incidence of these tumors has been variously estimated, from 0.2 per cent in a group of 487 autopsied brain tumors, by Frank,[8] to 0.66 per cent in Foerster's group of 750 tumors verified surgically.[12] In Cushing's group of 2209 cases from the Johns Hopkins and Peter Bent Brigham Hospitals, it was 0.6 per cent.[12] In our series, the incidence was 22 cases in 1952, or 1 per cent. There were 8 males and 14 females, while the ages ranged from 11 to 60 years inclusive.

TABLE 1

Comparison of the Surgical Treatment of Intradural and Extradural Epidermoids

	Total No. Tumors	Average Duration Preop. Symptoms	Complete Removal	Partial Removal	No. Reop.	Deaths
Extradural	7	2.9 years	4	3	2	0
Intradural	15	4.6 years	4	11	0	5

We have arbitrarily retained the name "epidermoid" as the term of preference and divided them into 2 groups, intradural and extradural (diploic), according to their surgical location. There were 15 intradural and 7 extradural tumors (Table 1).

SYMPTOMATOLOGY AND LOCATION

Headache was the predominant symptom in 14 of these patients and apparently was not related to whether the tumor was intra- or extradural in character.

Next to headache, visual disturbances were the commonest complaints. These consisted of blurred vision, diplopia, or actual blindness, and were found in 8 patients. Two of these were in the extradural group. Papilledema or optic atrophy was found in 11 patients, only 1 of these being in the extradural group. In 3 patients with extradural epidermoid, the predominant complaint was a small lump on the head, and in 2 of these the lump was tender. Among the patients with intradural tumors, convulsions were noted by 4, and 3 others complained of syncopal attacks.

The duration of symptoms varied from 6 months to 18 years. However, in the extradural group the average duration was 2.9 years compared to 4.6 years in the intradural group (Table 1).

Other authors have described the cerebellopontine angle to be the commonest site of occurrence.[3,10,18] In Mahoney's[12] group of 142 cases, collected from the literature, 112 were intradural. They occurred as follows: cerebellopontine angle—53, parapituitary—44, and 4th ventricle—15. As shown in Table 2, the incidence in our series was slightly different. Possibly, as our group is enlarged, the percentage of cerebellopontine angle lesions may increase.

TABLE 2

Location of Intradural Epidermoid Tumors

Cerebellopontine Angle	4
Parapituitary	4
Third Ventricle	2
Cerebellar Hemisphere	2
Cerebral Hemisphere	2
Vermis	1

DIAGNOSIS

Preoperatively all 7 of the extradural epidermoids were diagnosed correctly by roentgenography. That these tumors give a typical roentgenological picture was first pointed out by Cushing, in 1922.[7] Their sharp punched-out character with frequently associated white border is almost pathognomonic. Usually the inner table is slightly more involved than the outer (Figs. 1, 2 and 3). King[10] stated, "Any other eroding lesion, regardless of its nature, produces a defect in which the margin is less sharply defined, more

FIGS. 1 and 2. Pre- and postoperative films of extradural epidermoid tumor (N. W.).

hazy, and may be fuzzy and soft." One tumor (W. H.), occurring in the left temporal area, was both extra- and intradural and, although classified as intradural, it showed a typical roentgenographic picture of erosion. The commonest mistake in differential diagnosis of an epidural epidermoid is to call it a sebaceous cyst. In 4 out of 11 extradural or diploic type tumors reported by King,[10] a previous diagnosis of sebaceous cyst had been made. This error could have been eliminated with the aid of a preliminary roentgenological study. The intradural group offer a much greater challenge in diagnosis. There is usually nothing to suggest their diagnosis as epidermoids. In the series reported here, 2 of the parasellar group gave signs of sella erosion; a temporal lobe epidermoid revealed calcification; and 2 of the angle tumors showed erosion and destruction of the temporal bone. All were diagnosed preoperatively as probable tumors but not as epidermoids. Reporting in 1936, Love and Kernohan[11] stated that no intradural epidermoid had ever been diagnosed preoperatively by its roentgenological appearance.

FIG. 3. Preoperative film of epidermoid tumor which was both extra- and intradural (W. H.).

When seen at operation, these tumors are readily identified by their pearly sheen and avascular character. The only tumor presenting any similar characteristics is the dermoid. These are essentially much more cystic than the epidermoids and contain hair or other derivatives of the mesodermal corium in addition to ectodermal structures.

Two typical cases of epidermoid tumor, 1 extra- and 1 intradural, are presented. Case 1, an intradural tumor, shows the slow development and the typical symptomatology of these tumors when located in the cerebellopontine angle. The diagnosis of "epidermoid" was made by the operator as soon as the tumor came into view.

CASE REPORTS

Case 1. K. M., a 27-year-old white female, was admitted to the University Hospital, Mar. 30, 1936, on the service of Dr. Charles H. Frazier. She had been in normal health until 1932 when she began to note spells of weakness and dizziness following the birth of twins. In April 1933 she awoke one night and discovered that the left side of her face was paralyzed. At this time she was 3 months pregnant. In February 1935, at Neurological O.P.D., a left facial palsy and beginning staggering gait were noted. In October 1935, in Neurology Dispensary, she staggered slightly to the right on walking, with slightly increased reflexes on the right side. An audiogram, done at this time, revealed marked impairment of hearing on the left. The Barany test showed a delayed response on the left. Roentgenograms of the skull revealed no abnormal findings. Since December 1935, the patient had noted double vision and pins-and-needles sensation in the left lower jaw. There was no history of headache or vomiting, and her only other complaint was an annoying ticking sound in the left ear. She was admitted as an angle tumor suspect.

Neurological Examination. The above findings were confirmed with the addition of a left corneal anesthesia, an absent gag reflex, loss of taste on the left side of the tongue, and weakness of the left 6th nerve.

Operation. On April 2, 1936, a left suboccipital craniectomy was carried out under avertin anesthesia. After opening the dura and retracting the left cerebellar hemisphere, a tumor readily came into view. It had a laminated white appearance and was of cheesy consistency. It extended from the upper end of the spinal cord, up beyond the tentorium, and disappeared beneath the temporal lobe, lying close to the brain stem. A complete gross removal of the tumor was done in the posterior fossa plus a small amount of the portion extending up into the middle fossa.

Postoperative Course. She made an uneventful recovery. The only factor marring her convalescence was the development of a corneal ulcer in the left eye, which gradually healed following a tarsorrhaphy. When last seen, May 19, 1948, she was having slight trouble with her vision and still had some staggering gait. Neurological examination revealed flat discs and a left 7th nerve weakness. She was still able to carry out her housework quite satisfactorily and, in view of the fact that she had no papilledema, it was felt that there had been no gross recurrence of her tumor to this date.

Although this tumor was only partially removed at the time of operation in 1936, this woman has been able to carry out her daily work and live a relatively normal life for the past 11 years. This case emphasizes the fact that following evacuation of the contents and partial removal of the capsule of these tumors, they are extremely slow to present symptoms or signs of recurrence.

A typical case of complete removal of an extradural type of epidermoid shows that, although these tumors may occupy an entirely extradural

position, they require a major surgical attack if a thorough removal is to be carried out.

Case 2. E. B., a 43-year-old white railroad brakeman, was admitted to the University Hospital, Dec. 15, 1946. In 1940, a brain tumor had been accidentally discovered following roentgenography of his skull for a minor head injury. The roentgenograms showed a large area of cystic osteitis in the right occipital bone. The lesion involved both tables and there was some extension across the midline near the base. There was irregular absorption of bone throughout the whole area

FIGS. 4 and 5. Preoperative A-P and lateral films of extradural occipital epidermoid (Case 2).

and the pineal gland appeared to be displaced forward (Figs. 4 and 5). The patient had no complaints at that time and surgery was withheld until symptomatology developed. On admission he gave a history of increased difficulty in walking and a tendency to stagger for 1½ years. For about 4 years, he had had intermittent frontal headaches which had gradually progressed in severity.

Examination. The patient had moderate dyssynergia and dysmetria in the right hand. Also, there was a moderate speech disturbance consisting mainly of "mushiness" of his words. With the exception of an irregular, palpable, hard mass in the right occipital area, the physical findings were negative.

Operation. On Dec. 12, 1946, a large right occipital bone flap was turned down, with extension of the midline limb of the flap down to the rim of the foramen magnum. The bone flap was turned down just above the lateral sinus, running as far as the midline and laterally to include the posterior part of the parietal lobe. Over the dural surface, the whole of the right cerebellar area and the right occipital pole were compressed by a thick waxy material characteristic of an epidermoid tumor. In places the tumor was more than 4 cm. thick. All of this waxy material was removed, weighing a total of 195 gm. However, a portion of the capsule was firmly adherent to the lateral sinus and the dura, and it was impossible to remove this *in toto.*

Postoperative Course. The patient made an uneventful recovery and, when last seen June 16, 1948 he showed slight ataxia and slight dysmetria of the right hand. He was able to write with his right hand and was going back to work for the railroad company.

Microscopically the structure of the epidermoids has been best described by Bailey[1] as four concentric layers of varying thickness: (1) Stratum Durum: An outer acellular layer responsible for the pearly sheen. (2) Stratum Granulosum: Cuboidal to stratified epithelium. Cells often contain keratohyaline granules within cytoplasm. (3) Stratum Fibrosum: Layers of homogeneous material with no evidence of cell boundaries. (4) Stratum Cellulosum: Makes up the bulk of the tumor and consists of a mass of polyhedral cells.

Bailey states that the stratum durum varies from 1 to 5 or 6 times the width of the stratum granulosum. The stratum granulosum is the most constant in thickness and varies from 2 to 4 layers of flattened cells (Fig. 6).

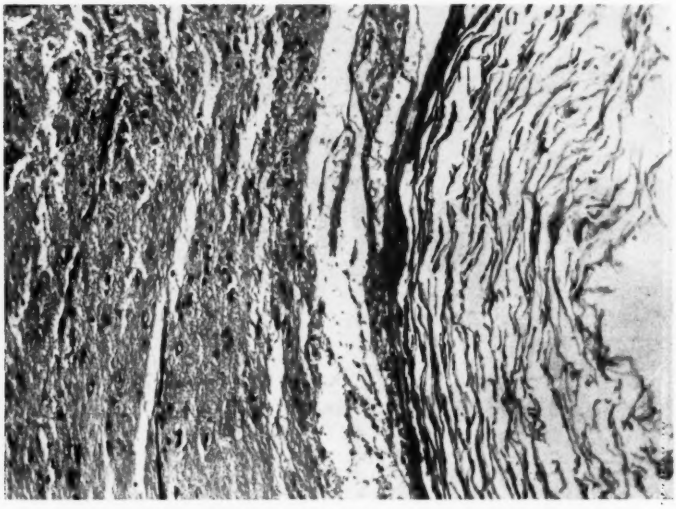

FIG. 6. Microscopic structure of typical epidermoid tumor (S. S.).

RESULTS

In only 8 of these 22 cases was a complete removal possible, and the weights of the tumors (complete and partial removals) varied from 13 to 195 gm. Four of the 15 intradural epidermoids could be completely removed: 1 in the posterior 3rd ventricle, 1 in the cerebellar hemisphere, and 2 in the left cerebral hemisphere. The patient with the 3rd ventricle tumor died 2 weeks postoperatively of a hemolytic Staph. aureus meningitis. The other 3 with complete intradural removals are alive and well, 11, 15, and 20 years postoperatively. Of the extradural group 4 out of 7 had complete removals.

No deaths occurred among the cases of extradural tumors. In the intradural group, 5 out of 15 died of the following causes: 2 hemolytic Staph. aureus meningitis; 1 respiratory failure on the table (tumor of vermis); 1 intraventricular clot; 1 bilateral atelectasis and pneumonia. In reviewing

these, it appears that the 2 deaths due to meningitis might conceivably have been avoided in these days of chemotherapy, since both patients were operated upon before 1935. Although most of the removals were partial in our series, only 2 reoperations were necessary, and these occurred in the extradural group. This emphasizes the slow growth of these tumors. Bailey,[2] and Rand and Reeves,[18] have both stated that the only true growing part of these tumors is the capsule. From this series it appears that if the bulk of the capsule is removed, one may expect a long period of quiescence before any signs of recurrence. Although the mortality rate is definitely higher in this benign group of tumors than in the meningiomas, there are several factors to consider. The intradural epidermoids are known to arise close to the midline, and this increases the difficulty of surgical removal. Due to their extremely slow growth, they usually fail to show signs of pressure until a far advanced stage. Finally, there were only 2 tumors of the convexity in this group (W. H. and H. B.), while in the meningiomas approximately 70 per cent occur in the convexity. These factors all tend to increase the relative mortality. Two conclusions stand forth. First, the extradural type should be completely removed at the first operation, if one is careful completely to circumscribe all of the involved bone. Second, the intradural group are frequently involved with vital midline structures including cranial nerves and carotid arteries. In view of their extremely slow growth and the fact that no reoperations were necessary in our series of cases, the surgeon should not always strive at total removal but be satisfied with complete evacuation and removal of as much of the capsule as possible.

SUMMARY

1. Twenty-two operative cases of epidermoid tumor of the brain have been reviewed with special reference to symptomatology, roentgenological findings, and preoperative diagnosis.

2. All of the 7 extradural epidermoids showed roentgen changes of the skull typical of this type of tumor. Of the 15 intradural tumors, 14 could not be diagnosed preoperatively by their roentgen appearance.

3. The extradural group of tumors showed a preoperative duration of symptoms averaging 2.9 years compared to an average duration of 4.6 years for the intradural group.

4. The commonest sites of occurrence, of the intradural group, were the cerebellopontine angle and the parapituitary region, with 4 occurring in each position.

5. Four of the 7 extradural and 4 of the 15 intradural tumors were completely removed with an over-all mortality of 22.7 per cent.

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THE SURGICAL TREATMENT OF ARNOLD-CHIARI MALFORMATION IN ADULTS

AN EXPLANATION OF ITS MECHANISM AND IMPORTANCE OF ENCEPHALOGRAPHY IN DIAGNOSIS

W. JAMES GARDNER, M.D., AND ROBERT J. GOODALL, M.D.
Cleveland Clinic, and the Frank E. Bunts Educational Institute, Cleveland, Ohio

(Received for publication August 12, 1949)

THE ARNOLD-CHIARI malformation is a deformity of the hindbrain in which a tongue-like projection of the cerebellar tonsils protrudes through the foramen magnum down onto the cervical cord. The caudal portion of the 4th ventricle likewise is elongated downward. The upper cervical nerve roots pursue a cephalad direction, giving the impression that the brain stem has been drawn down through the foramen magnum.

The malformation was named after Arnold[3] and Chiari[6] who independently described the anomaly in 1894 and 1895 respectively. Schwalbe and Gredig[23] published an embryologic and anatomic treatise on the subject in 1907.

We have operated upon 17 patients, adults and adolescents, with the Arnold-Chiari malformation and our experience suggests that the fundamental mechanism is obstructive hydrocephalus with resulting foraminal herniation of the hindbrain.

Interest in the anomaly was revived in 1935 when Russell and Donald[22] discussed its importance as a possible cause of internal hydrocephalus in spina bifida. They presented 10 examples of myelomeningocele associated with hydrocephalus and Arnold-Chiari malformation in infants. McConnell and Parker[16] reported surgical treatment of 5 patients ranging in age from 10 to 32 years with 2 successful results. All were found to have some degree of hydrocephalus. D'Errico[8] in 1939 described 10 cases of myelomeningocele with hydrocephalus in infants. The myelomeningocele was repaired in 7, and when hydrocephalus followed, operation for the Arnold-Chiari malformation was done. Three remained in good health for a period of 2 years thereafter. He stressed the advisability of doing a suboccipital decompression in all cases of spina bifida associated with myelomeningocele and hydrocephalus. In 1939, McKissock[17] stated that he routinely treated hydrocephalus with associated myelomeningocele by first decompressing the Arnold-Chiari malformation, and that this frequently benefited both the hydrocephalus and the sac protrusion.

Chamberlain[5] in 1939 described concomitant findings of Arnold-Chiari malformation with platybasia and described the latter condition as follows: "The morphologic changes shown by roentgenograms give the impression of softening of the base of the skull and moulding through the force of gravity. It is as though the weight of the head has caused the ears to approach the

shoulders, while the cervical spine, refusing to be shortened, has pushed the floor of the posterior fossa upward into the brain space." Gustafson and Oldberg[10] in 1940 called attention to the relationship between Arnold-Chiari malformation, Klippel-Feil syndrome, syringomyelia and platybasia. They pointed out the possibility of benefiting syringomyelia by surgically unblocking the fluid pathways between the cranial and spinal cavities.

List[15] in 1941, in a scholarly discussion of neurologic syndromes accompanying developmental anomalies of the occipital bone, atlas and axis, reported 3 cases of Arnold-Chiari malformation operated upon successfully. He refers to the foraminal herniation of cerebellar tissue as Arnold's deformity, while the associated posterior bulging or kinking of the medulla he calls Chiari's deformity. He stresses the importance of posterior dislocation of the dens as a factor in the production of neurologic signs.

Ogryzlo[19] in 1942 reported 7 operative cases, 4 in infants associated with spina bifida. Three were in adults, of which 2 improved after operation and 1 died. He suggested that hydrocephalus might be responsible for the Arnold-Chiari deformity and that the hydromyelia could be the result of fluid being forced down the central canal.

Ray[21] in 1942 described 2 cases of congenital and 2 of acquired platybasia with involvement of the central nervous system. He also commented upon the similarity of the appearance of the brain and cord in cases of platybasia and in Arnold-Chiari malformation.

Lichtenstein[13,14] in 1943 likewise called attention to the similarity of the appearance of the cerebellar tonsils in platybasia and in Arnold-Chiari malformation. He believes that the accumulating cerebrospinal fluid in these cases may dilate the central canal with resulting hydromyelia and may also perforate the ependymal lining with the formation of a syrinx.

Ingraham and Scott[11] in 1943 presented an excellent review of the literature and described 20 cases of Arnold-Chiari malformation in infants associated with myelomeningocele. The majority of their patients had associated microgyria and craniolacunia but only 2 had platybasia.

There are several other case reports of this interesting malformation, including those of Aring,[2] Penfield and Coburn,[20] Adams, Schatzki, and Scoville,[1] Ecker and Ferguson,[9] Shryock,[24] Wagner,[26] Moore,[18] Bucy and Lichtenstein,[4] Steele,[25] and Chorobski.[7]

We have reviewed the cases of Arnold-Chiari malformation reported in the literature and, exclusive of infants, found 17 cases verified and treated surgically. There are several other probable cases classified under other diagnoses, particularly platybasia.

Exclusive of infants, the files of the Cleveland Clinic Hospital of the last 10 years contain the records of 17 patients with Arnold-Chiari malformation who were treated surgically. The ages of these patients varied from 14 to 54 years. The mean duration of symptoms was 3 years, but it varied from 26 years to 7 months. The onset of symptoms followed a febrile illness in 2 cases and a fall in 2 cases. In the remaining 13 instances there was no

history of a factor that precipitated the symptoms. The complaint that the patients stressed was weakness and/or numbness of one or more extremities in 12 cases, ringing in the ears and loss of hearing in 2 cases, headache, girdle pain, and difficulty in breathing in 1 case each. Other complaints proffered by these patients consisted of double vision, loss of balance, pain in the arm, curvature of the spine, incontinence of urine, hoarseness, difficulty in swallowing, and ulceration of the finger tips.

The most common finding was weakness of one or more extremities; in only 2 of the 17 cases this was not demonstrable. It involved the upper more than the lower extremity. There were 5 cases of hemiparesis and 3 of quadriparesis. In 1 case the weakness involved both legs and in another one leg. There was a syringomyelic type of sensory loss in 12 cases, although in 2 of these, no cyst was found at operation.* The sensory loss included the 2nd cervical dermatome in 10 of the cases. Ten patients exhibited nystagmus, and the Romberg test was positive in 7. Two patients had astereognosis. In only 1 case was there papilledema. The preoperative diagnoses in these 17 cases were cord tumor, 3; platybasia, 2; acoustic tumor, 2; syringomyelia, 2; brain tumor, 1; multiple sclerosis, 1. The last 6 cases were observed within a period of 6 months and the preoperative diagnosis of Arnold-Chiari malformation was made correctly in each instance.

A careful review of the roentgenograms in these 17 cases disclosed definite evidence of platybasia in 8 (Fig. 1). In these 8 cases the tip of the dens projected above Chamberlain's line† for from 6 to 30 mm. In 3 additional cases the appearance suggested a mild degree of platybasia while in only 4 instances could the x-rays of the skull and cervical spine be said to be entirely normal. There were 2 cases of Klippel-Feil syndrome and 1 of hemivertebra at the level of the 12th dorsal and 1st lumbar vertebrae. In 8 of these 17 cases, there was some degree of scoliosis of the dorsal spine. In none of these patients were there any physical signs suggesting an occult spina bifida although in 2 cases a failure of fusion of the 5th lumbar arch was demonstrated by x-ray.

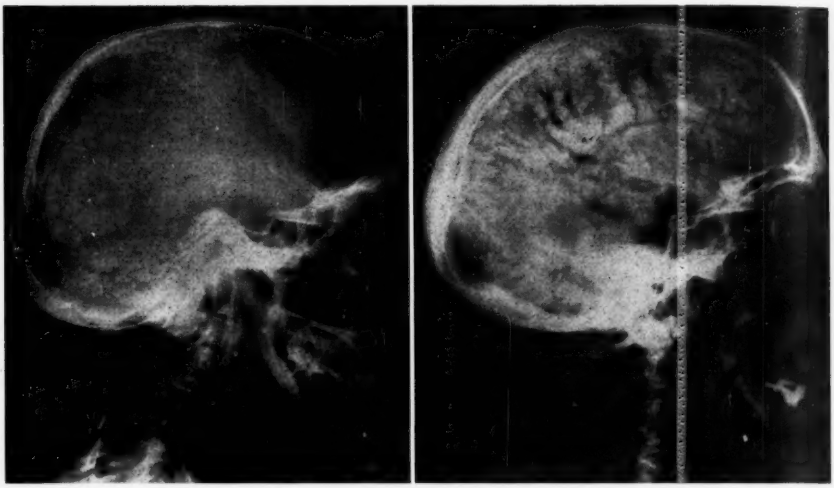
In 3 of these 17 cases a subarachnoid block was demonstrated at spinal puncture but there was no increase in the total protein content. In 2 instances without subarachnoid block, the total protein content of the spinal fluid was elevated. Pneumo-encephalography was carried out in 11 cases and little or no air entered the ventricles (Fig. 2). The shape of the corpus callosum and the lateral ventricles, when outlined, indicated the presence of hydrocephalus. The pontine cisterns were flattened and air could not be demonstrated in the cisterna magna. In 1 case in which a cisternal pantopaque injection was attempted, the oil was found on x-ray to have been introduced into the caudally displaced 4th ventricle.

In the majority of these cases the symptoms began during adulthood.

* Of the 13 patients who had cystic spinal cords at operation, 3 exhibited no sensory loss.

† Chamberlain's line: A line drawn between the hard palate and the posterior rim of the foramen magnum.

A possible explanation of this late development of signs in cases of a congenital disorder is as follows. Mild and unrecognized hydrocephalus with Arnold-Chiari deformity is present in infancy, but becomes compensated so that the growth of the brain catches up with that of the skull. Thus, when adulthood is attained, the circumference of the skull may be normal, although some degree of platybasia may be present. Then, due to the increasing density of aging connecting tissues, aggravated perhaps by infection or



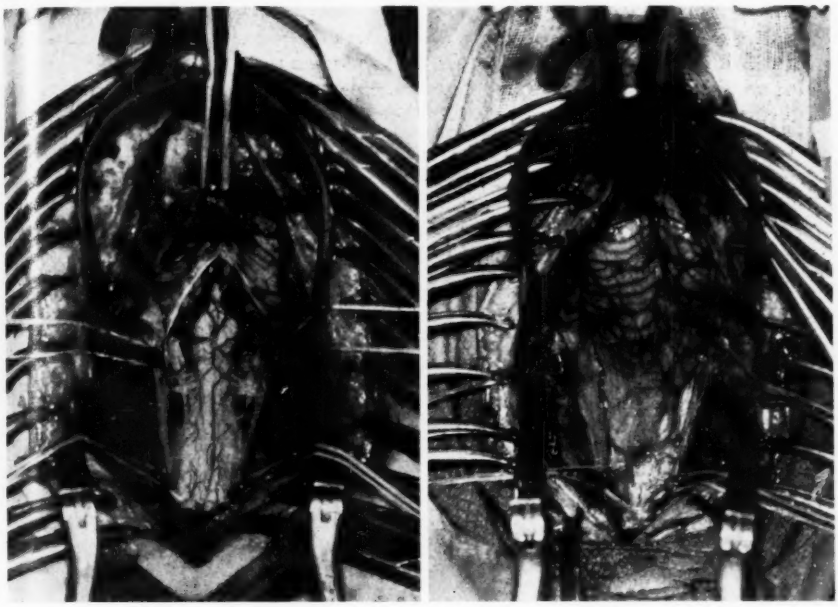
FIGS. 1. and 2. *Left,* A case of advanced platybasia with Klippel-Feil syndrome and operative findings of Arnold-Chiari malformation. *Right,* Encephalogram in a typical case of Arnold-Chiari malformation.

trauma, the outlets of the 4th ventricle become less pervious to fluid, so that an increased head of pressure is required to filter the fluid through to the subarachnoid cisterna. The resulting progression in the foraminal herniation is responsible for the gradually developing symptoms.

SURGICAL TREATMENT

These patients were operated upon in the sitting position. Through a midline incision, an opening was made in the occipital bone including the posterior rim of the foramen magnum. The laminae of the upper cervical vertebrae were removed. As the dura was incised, the cerebellar tonsils were found to fill the cisterna magna and to extend through the foramen magnum for a variable distance. The dura was usually adherent to a thickened arachnoid membrane at the level of the foramen magnum. The foramen of Magendie was closed by a dense membrane or by adhesion of the cerebellar tonsils to each other. The 2nd cervical nerve roots pursued a cephalad course

to their points of exit from the dural sac.* The exposed portion of the cervical cord was cystic in 13 instances and in each case the aspirated fluid was clear and colorless. In 2 cases the total protein content of the cyst fluid was estimated and found to be identical with that in the spinal fluid. An attempt was made to reopen the foramen of Magendie in each case and the dura, of course, was left open (Figs. 3, 4, and 5).



FIGS. 3 and 4. *Left*, Operative exposure illustrating for the sake of comparison a normal brain stem. *Right*, A 44-year-old woman with progressive 8th nerve and cerebellar symptoms for many years. The tip of the left cerebellar tonsil reached the level of the 3rd cervical lamina.

In this series of 17 patients, 13 were definitely benefited by the operation, 3 were made worse because of aggravation of a quadriparesis, and 1 died 18 hours after operation of respiratory failure. In retrospect we believe that postoperative aggravation of symptoms may be due to a squeezing of the cord produced by flexion of the head during the positioning of the patient for operation.

A CASE OF ACQUIRED ARNOLD-CHIARI MALFORMATION

The following additional case of an Arnold-Chiari malformation, sub-

* This stretching of C-2 is probably responsible for the occipital headache so frequently complained of by these patients and it may explain the fact that a pin prick usually feels less sharp on the posterior portion of the scalp than on the anterior.

sequently proved to be due to an acoustic tumor, offers some interesting ground for speculation in regard to the mechanics of hydrocephalus with associated hydromyelia.

The patient was a woman of 50 who presented herself on Mar. 29, 1945 because of headaches of 4 months' duration. She exhibited bilateral papilledema, complete deafness in the right ear with absence of caloric response, and partial deafness in the left ear. There were definite cerebellar signs and impairment of sensation in the distribution of both 5th nerves. An acoustic tumor was diagnosed, but when spinal puncture disclosed normal pressure with complete spinal subarachnoid block the diagnosis was changed to tumor at the foramen magnum.

A midline exposure of the cerebellum was carried out, together with laminectomy of the upper four cervical vertebrae. As the dura was opened, the cervical cord was

FIG. 5. A 44-year-old woman with progressive weakness and numbness of arms for 2 years. The cerebellar tonsils are protruding through the foramen magnum, the 2nd cervical nerve roots pursue a cephalad course, there is a posterior bulging of the medulla (Chiari deformity), and below that a cystic dilatation of the cervical cord.

found to be distended with fluid. There was a pronounced herniation of the cerebellar tonsils. A needle was introduced into the spinal cord and clear, colorless fluid was aspirated. An incision was made into the hydromyelic cyst, after which the distended cord collapsed. The cerebellar tonsils were separated by retractors and the floor of the 4th ventricle exposed. It was deformed by a firm swelling at the level of the calamus scriptorius. This led the surgeon to suspect a tumor of the medulla with associated hydromyelia. The course of the 2nd cervical nerve roots was not described.

The patient improved, but 14 months following this operation, she returned with unmistakable signs of a right 8th nerve tumor. She was operated upon and a typical acoustic tumor was found and removed. The patient again improved, but died suddenly 5 months later, presumably of a cerebral vascular accident. Postmortem examination was not obtained.

In retrospect we believe that the hydromyelia in this case, as in the case

of congenital Arnold-Chiari malformation, was the result of extrusion of ventricular fluid into the patent central canal of the cord from the obstructed 4th ventricle, and that the swelling of the medulla was due to a crowding downward of the brain stem (Chiari deformity). We believe that hydromyelia is commonly associated with the congenital form of Arnold-Chiari malformation and but rarely seen in the acquired form produced by cerebellar tumor because, as pointed out by Kramer,[12] the central canal is always patent in infancy and relatively rarely so in the adult.

CONCLUSIONS

Although fully cognizant of the assailability of deductions drawn from an experience which has been almost purely surgical, nevertheless the following conclusions seem inescapable.

1. The Arnold-Chiari malformation is merely a foraminal herniation of intracranial structures produced by an obstructive hydrocephalus. Cord traction resulting from myelomeningocele, when present, may contribute to the deformity. The associated hydromyelia is due to the extrusion of fluid from the obstructed 4th ventricle downward into the central canal of the cord. A descriptive term for the condition is congenital obstructive hydrocephalomyelia with foraminal hernia.

2. The foraminal hernia which we call Arnold-Chiari malformation differs from the foraminal hernia produced by a cerebellar tumor because the former is a more chronic condition, produced by a relatively small increase in intraventricular pressure, which has its beginning during intrauterine life.

3. The "congenital" form of platybasia is produced by the increased weight of the head and malleability of the skull bones in cases of congenital obstructive hydrocephalus. On the other hand, if the infant is unable to assume the erect posture because of an associated myelomeningocele, platybasia should not develop.

4. In acquired platybasia of Paget's disease, osteogenesis imperfecta, and other bone-softening disorders, an Arnold-Chiari-like deformity results from the extrusion of the hindbrain through the foramen magnum due to the encroachment on the volume of the brain case occasioned by the deformity. While this may resemble the congenital variety of true Arnold-Chiari deformity it is not accompanied by hydrocephalomyelia.

5. Arnold-Chiari malformation should be suspected in any patient with signs suggesting syringomyelia, syringobulbia, platybasia, Klippel-Feil syndrome, and also in cases of unexplained scoliosis. It should be particularly suspected in the above cases if cerebellar signs are present or if there is impairment of pain perception in the distribution of the 2nd cervical nerve.

6. The diagnosis may be confirmed in the above cases by encephalography. The films will disclose evidence of obstructive hydrocephalus with absence of air in the cisterna magna. Since the intraventricular pressure is

usually only slightly increased, this procedure is safe provided the surgeon is prepared to operate at once if the films confirm the diagnosis.

7. Surgical treatment should be aimed at relieving the obstructive hydrocephalus.

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THE ELECTROENCEPHALOGRAM IN SUBDURAL HEMATOMA

WITH A REVIEW OF THE LITERATURE AND THE PRESENTATION OF SEVEN CASES*

GEORGE W. SMITH, M.D.,† WILLIAM H. MOSBERG, M.D.,†
E. T. PFEIL, M.D.,† AND R. H. OSTER, PH.D.‡

Department of Neurological Surgery, University of Maryland, Baltimore, Maryland

(Received for publication August 26, 1949)

THE PATHOLOGY and treatment of subdural hematoma is well understood but the clinical picture is subject to great variance. The writers are impressed, both in clinical experience and in the review of the literature, by the difficulty with which an early diagnosis is made.[4,6] Most publications on subdural hematoma are prefaced by statements that emphasize the inconsistent symptomatology and difficulty in diagnosis. It is the attitude of some surgeons that whenever subdural hematoma is suspected a simple and expedient method of proof is to perform bilateral trephines.[3] With such a routine the percentage of negative explorations increases. It is desirable to place the trephine openings in only those patients having a surgical lesion. This feeling gains added strength since trephine openings have an implication in the patient's industrial, insurance and psychic future.

For these reasons additional diagnostic procedures have been employed. Pneumoencephalography is not without risk and is considered contraindicated by some authorities.[9] In the study of subdural hematoma, as in all neurological disease, diagnostic tests have been directed mainly toward disturbances of anatomical relations. With the confirmation of Berger's observation that the electrical activity of the brain followed a certain rhythmical pattern, and that in pathological states this fundamental pattern was altered, a useful diagnostic aid was placed in the hands of the clinician. The electroencephalograph offers a new and different approach in that alteration in the *physiologic* (electric) *activity* of the brain can be studied directly as well as indirectly. With subdural hematoma the abnormalities in electrical activity of the brain are shown to be altered dependent on: (1) the amount of the bleeding, (2) the location, and (3) the time interval between the bleeding and the EEG.

There are reported in the literature several clinical and experimental observations on the EEG in subdural clot that appear conflicting. The EEG has been described as showing (1) increased voltage and slow wave pattern

* Aided by a grant from the Hoffberger Neurosurgical Fund, University Hospital, Baltimore, Md.
Read before the Society of Neurological Surgeons, June 10, 1949, Baltimore, Md.

† Department of Neurological Surgery.

‡ Department of Physiology, School of Dentistry.

on the side of the lesion; (2) decreased voltage and slow wave pattern on the side of the lesion with normal pattern on the contralateral side; 3) decreased voltage and slow wave pattern on the side of the hemorrhage with increased voltage and fast wave activity on the contralateral side; or 4) normal electrical activity.

Walter in 1939,[14] who was the first to report focal abnormal electrical activity of the brain with subdural hematoma, concluded that a "silent area" existed over the site of the hematoma. He apparently used the term "silent area" as meaning an area of decreased electrical activity (amplitude).

Cohn,[1] in a carefully executed experiment with cats, produced subdural hemorrhages by instilling the animals' own blood into the subdural space. He was able to produce varied EEG findings by taking recordings at intervals up to 21 days. The abnormal electrical activity of the brain in these animals correlated directly with the extent and duration of the hemorrhage, which in turn caused varying degrees of injury to the cortical neurones. With the experimentally produced subdural hematoma there was decreased electrical activity over the site of the hematoma after the first 30 minutes, which persisted for at least 1 week. He found that subdural hematoma of 1 week or more in duration may show: (1) *focal decreased activity* (amplitude) over the site of the hematoma; (2) generalized slow waves with a focus of abnormality; or (3) the tracing may be normal.

Cohn *et al.*,[2] in reporting 6 clinical cases, stated that the EEG showed one of two primary characteristics; a maximum of high voltage slow waves over the lesion (3 cases) or slow wave activity of reduced amplitude over the site of the disturbance (2 cases). He was unable to explain the absence of electrical changes in 1 case of surgically proved subdural hematoma.

Gurdjian and Webster,[5] in their article on intracranial hemorrhage, commented that the EEG may be of important assistance in the diagnosis of subdural hematoma and stated that correct localization had been accomplished by this technique in 40 per cent of their cases. They did not describe the criteria used for localization except to state that their records showed "disorganization of electrical pattern on the affected side." Of the 3 tracings that they showed, each case appeared to us to show *decreased amplitude* over the hemisphere underlying the hematoma. They failed to comment on this amplitude variation.

Ulett[13] produced subdural hematomas in dogs experimentally and concluded that decreased voltage occurred over the site of the hematoma with no changes being observed over the contralateral side. This abnormality persisted until the dogs were killed 2 days later.

Heersema and Freeman[6] reported that of 25 cases of subdural hematoma the EEG was helpful in one-half and disclosed unilateral delta waves. They stated that the delta waves were so extensive in some instances that the EEG was not an aid to localizing the lesion. In 12 per cent of their cases the EEG findings were normal but these were considered erroneous. From their article it is impossible to draw any definite conclusions. They failed to give

the electrode locations, the type of tracings, the duration of the hematoma prior to the EEG and the extent of the hematoma over the hemisphere as shown at surgery or autopsy. They made no mention of amplitude variations.

Sjaardema and Glaser,[12] in a report of 3 verified cases of subdural hematoma, concluded that the EEG pattern consisted of high voltage beta activity combined with delta waves. They felt this pattern was not pathognomonic of subdural hematoma since it had been observed in other pathological states. Careful analysis of their cases and of tracings in their Case 3 (the patient expired) showed the brain to have sustained multiple severe injuries. We conclude that their EEG was not representative of a "pure" subdural pattern. In their Case 2 the tracing presented in Fig. 2 appears to the writers to show *decreased amplitude* over the site of the hematoma. The representative tracing in their Case 1 was filled with artifacts and no conclusion seemed apparent.

Rogers,[11] in a single case report of chronic subdural hematoma, showed the EGG to be characterized by a "very low potential discharge at varying frequencies in the right fronto-temporal region, most definite in the neighborhood of the Sylvian point." A subtemporal craniotomy revealed a chronic subdural hematoma in that area.

Jasper *et al.*[7] found that with a subdural collection of fluid the EEG showed decreased amplitude over the site of the hematoma when a bipolar tracing was taken with the recording electrodes placed *over* the hematoma and not adjacent to it. Their observations were based on clinical cases. In a later article[8] they concluded that an asymmetry in amplitude (with the decreased amplitude being over the hemisphere of the hematoma) of simultaneous records from bilaterally homologous regions was the most reliable localizing criterion.

The following 7 proved cases of unilateral subdural hematoma show EEG patterns that correlate with the operative findings. Following the surgical removal of the hematomas the abnormal EEGs progressively improved and in the 6 surviving patients returned to normal over a period of 1 to 5 months.

CASE REPORTS

Case 1. W.B., a white male aged 36, was admitted to the University Hospital in a moderate stupor on Nov. 16, 1947. On Nov. 1, 1947 he had been found unconscious on the street after allegedly having been struck by an auto. He regained consciousness 48 hours after injury and was discharged from a county hospital ambulatory on the 10th day. He was admitted to this hospital 16 days after injury because of headache, nausea, vomiting and stupor.

Examination. He had pulse 64, respirations 30, bilateral early papilledema, dilated and fixed *right* pupil, right hemiparesis, and unsustained ankle clonus on the right. Lumbar puncture: initial pressure 140 mm. water; xanthochromic CSF.; traces of globulin.

Operation. A chronic encapsulated liquid subdural hematoma on the right was evacuated through a right temporoparietal trephine. Exploratory trephine on the left revealed no intracranial pathological process.

Course. He was discharged asymptomatic 10 days following operation.

EEG. The pre-operative EEG (Fig. 1), bipolar potentials, was characterized by a 75 per cent reduction in amplitude over the entire right hemisphere with low random, low voltage 1–2/sec. waves. An underlying 22/sec. spiky low-voltage pattern was seen generalized. Repeated tracings (Fig. 2) showed a gradual return to what is thought to be the normal pattern for this person.

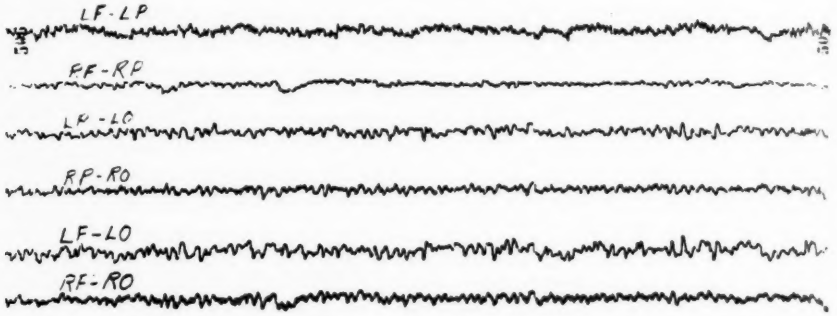

FIG. 1. Case 1. Pre-operative record. Note the great lowering of amplitude over the entire right hemisphere, particularly RF-RP.

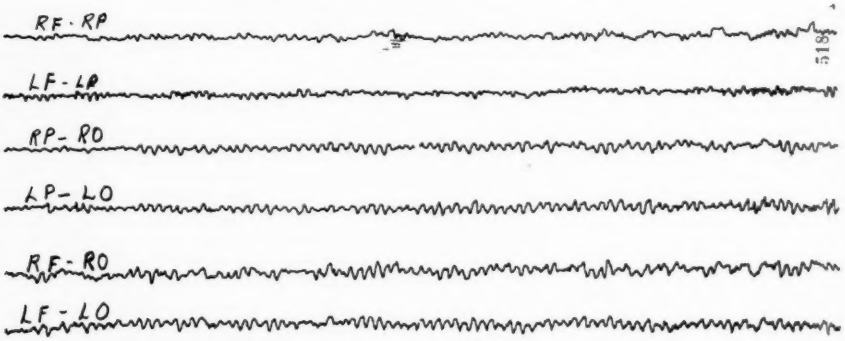

FIG. 2. Case 1. Tracing 5 months postoperative—thought to be normal for this individual. The amplitude over the right hemisphere now equals that over the left.

Case 2. L.M., a white female aged 59, was admitted Sept. 25, 1948. She had had frequent headaches since childhood and forgetfulness for the last 2 months. Three weeks before admission she fell down a flight of stairs. Ten days later she complained of severe headache, went into coma and was transferred to this hospital.

Examination. Temperature, pulse and respirations were normal. The *left* pupil was slightly larger than the right. Bilateral Babinski response to plantar stimulation was elicited. There was right hemiparesis, with hyperactive deep tendon reflexes on the right.

Operation. An encapsulated liquid subdural hematoma on the right was evacuated through a right temporoparietal trephine. Exploratory trephine on the left revealed no evidence of an intracranial pathological process.

Course. Convalescence was uneventful and she was discharged asymptomatic 16 days after operation.

EEG. The pre-operative EEG (Fig. 3) showed a 50 per cent reduction in amplitude over the right fronto-temporo-parietal region during the bipolar potential tracing. Irregular 4–6/sec. sharp waves were seen mainly over the right hemisphere. A repeat tracing 2 weeks later (Fig. 4) showed a generalized 20/sec. low voltage spiky pattern with no obvious asymmetry in amplitude.

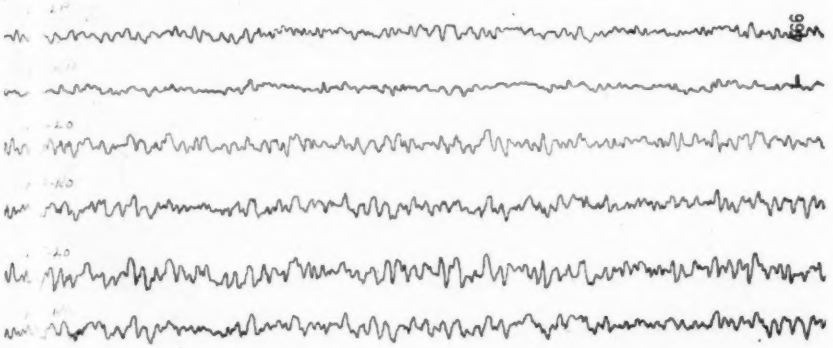

FIG. 3. Case 2. Pre-operative tracing. Note the great reduction in amplitude over RF-RP areas (channel 2). Generalized multiple sharp and spike waves are present.

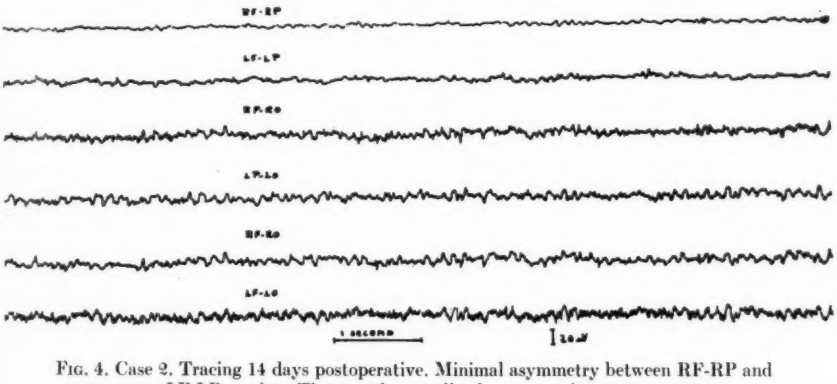

FIG. 4. Case 2. Tracing 14 days postoperative. Minimal asymmetry between RF-RP and LF-LP persists. The record generally shows great improvement.

Case 3. S.J., a colored female aged 40, was admitted Oct. 25, 1948. Three weeks previously she had been struck on the right side of the head. Two weeks after injury slurred speech, drowsiness, weakness of right side of body and blurring of vision developed.

Examination. Temperature, pulse and respirations were normal. She had a right hemiparesis, hyperactive deep tendon reflexes on *right*, Babinski response to plantar stimulation on right, and dilated *right* pupil.

Operation. There was an encapsulated liquid subdural hematoma over the left hemisphere. Exploratory trephine on the right disclosed no evidence of an intracranial pathological process.

Course. She was discharged asymptomatic 17 days after the operation.

EEG. The pre-operative EEG (Fig. 5) was characterized by a 50 per cent decreased amplitude over the left hemisphere, particularly over the left frontoparietal area. Generalized sharp waves and spike forms were seen over the right hemisphere. Only occasional random 1/sec. waves were seen over the left hemisphere. A repeat tracing (Fig. 6) 1 month postoperative showed no appreciable amplitude variation.

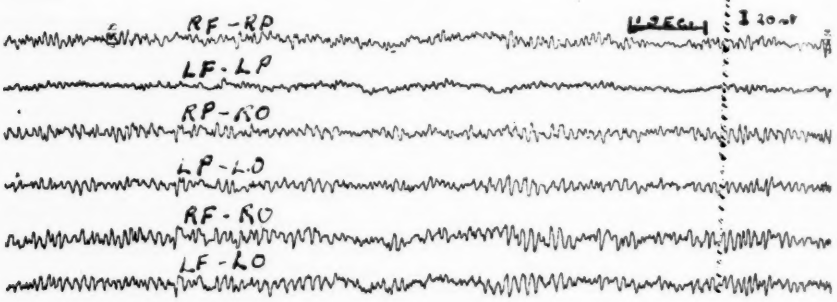

FIG. 5. Case 3. Pre-operative tracing. Note lowered amplitude over the left hemisphere, particularly the left frontoparietal region.

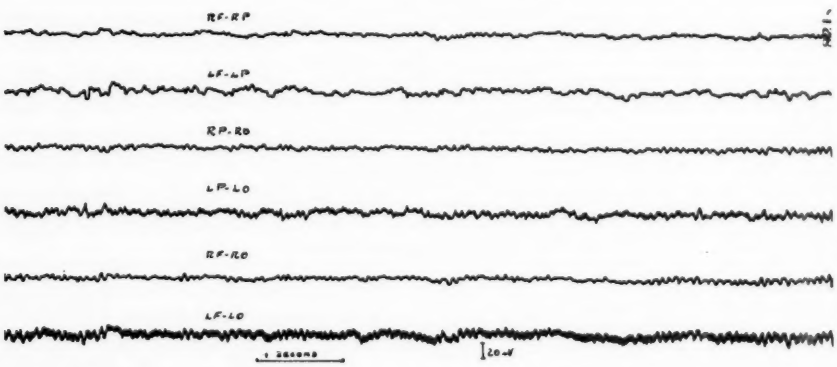

FIG. 6. Case 3. Tracing 1 month postoperative. Note that the amplitude variation between hemispheres is no longer present. Random slow waves persist over the left hemisphere.

Case 4. J.H., a colored male aged 37, was admitted on Nov. 9, 1948. He had been struck on the left side of the head with a bottle 8 days previously. He had complained of headache and lethargy, and 8 days after injury weakness of the right upper and lower extremities developed and he then became stuporous.

Examination revealed pulse 60, stupor, right hemiparesis, and right central facial weakness.

Operation. An encapsulated liquid subdural hematoma on the right was evacuated through a right temporoparietal trephine opening. Exploratory trephine on the left revealed no evidence of an intracranial pathological process.

Course. He was discharged asymptomatic 7 days after operation.

EEG. The pre-operative EEG (Fig. 7), with bipolar potentials, was char ac-

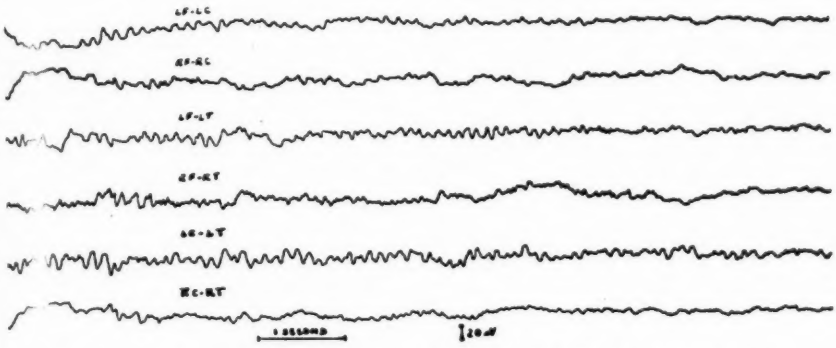

FIG. 7. Case 4. Pre-operative tracing. Note marked lowering of amplitude over entire right hemisphere. Random low voltage slow waves are seen over the right hemisphere.

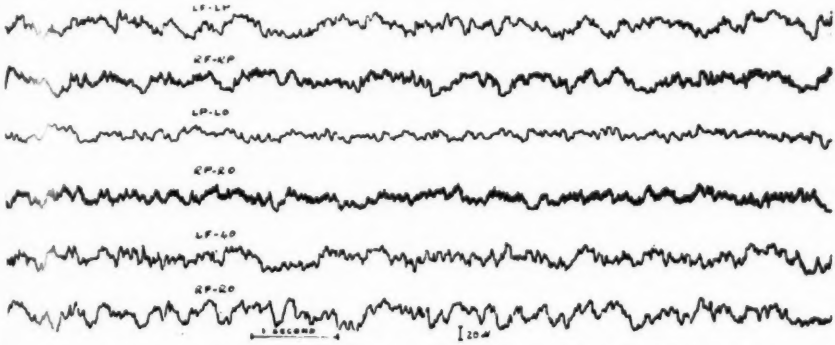

FIG. 8. Case 4. Tracing 4 days postoperative. The asymmetry of amplitude between hemispheres is minimal. Generalized multiple high voltage slow waves are now present.

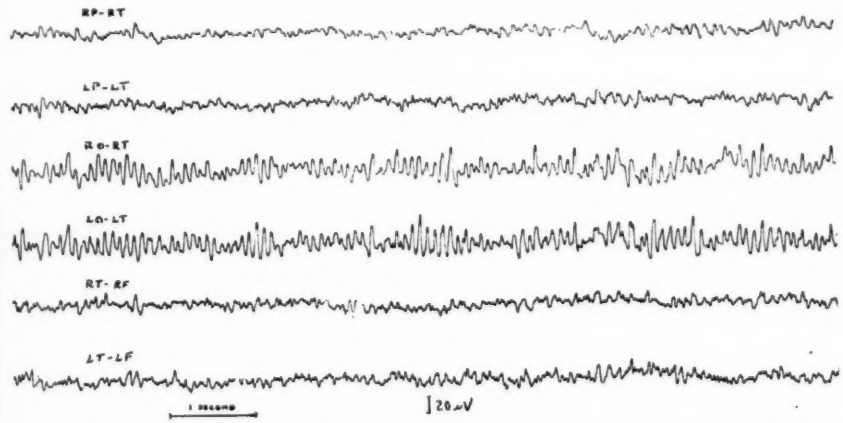

FIG. 9. Case 4. Tracing 6 months postoperative. The asymmetry of amplitude between hemispheres is no longer present, and the generalized multiple high voltage slow waves have disappeared. The pattern shows random and multiple sharp waves of increased voltage and random spike forms.

terized by a 60 per cent reduction of amplitude over the right fronto-temporo-parietal area. Random sharp waves were present generally with occasional 2/sec. slow waves over the right hemisphere. A tracing 4 days postoperative (Fig. 8) showed only minimal lowering of amplitude over the right hemisphere with multiple high voltage slow waves. Six months postoperative (Fig. 9) the multiple high voltage slow waves disappeared and random sharp waves of increased voltage and spike forms were present.

Case 5. E.B., a colored male aged 54, was admitted on Dec. 21, 1948, complaining of numbness and weakness in the right upper, and to a lesser extent, right lower extremities. Two weeks previously he had been struck on the head in a fight. Three days prior to admission he had complained of headache and became confused, and on the last day he had a convulsive seizure (details not known).

Examination. Pulse was 52, and blood pressure 200/120. He was confused and lethargic. He had dilated *right* pupil, right central facial weakness, right hemi-

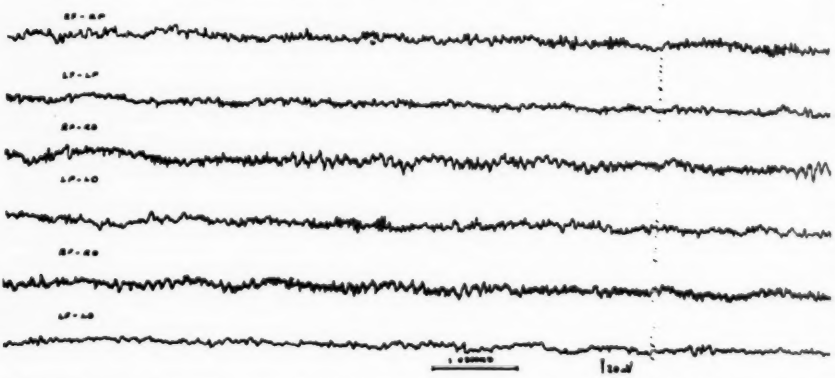

FIG. 10. Case 5. Pre-operative record. Note the marked lowering of amplitude over the left hemisphere, most particularly the LF-LO leads. Minimal random slow waves are seen.

paresis, and loss of position sense and stereognostic sense in the right upper extremity.

Operation. A chronic encapsulated liquid subdural hematoma on the left was evacuated through a left temporoparietal trephine. Exploratory trephine on the right revealed no intracranial pathological process.

Course. He was discharged asymptomatic 14 days after operation.

EEG. The pre-operative tracing (Fig. 10) was characterized by a 50 per cent decrease in amplitude over the left hemisphere. Random 1–2/sec. waves of increased voltage are superimposed on a fundamental frequency of 10/sec. waves.

A tracing taken immediately after operation (Fig. 11) showed no appreciable amplitude difference. The 1–2/sec. waves were replaced by random 3–4/sec. slow waves. A repeat tracing 3 weeks postoperatively (Fig. 12) showed the pattern to be approaching normal.

Case 6. M.R., a white female aged 45, was admitted Feb. 21, 1949. One hour before admission she had fallen on the sidewalk, and lapsed into profound coma.

Examination. Pulse was 60. She had absent corneal reflexes, flaccid paralysis of all extremities, most marked on left, left central facial weakness and a dilated *right* pupil. Within ½ hour both pupils became dilated. Bloody CSF was oozing from the left ear. Roentgenograms of skull showed a linear fracture of the left frontotemporal region.

Operation was performed immediately and a massive subdural hematoma, 4 cm.

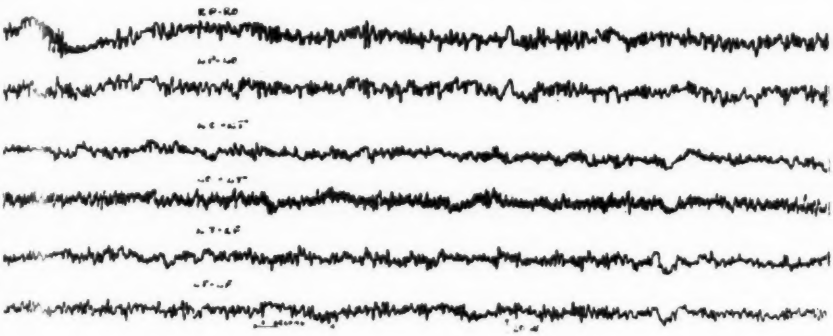

FIG. 11. Case 5. Tracing 1 hr. postoperative. The amplitude asymmetry between hemispheres has disappeared.

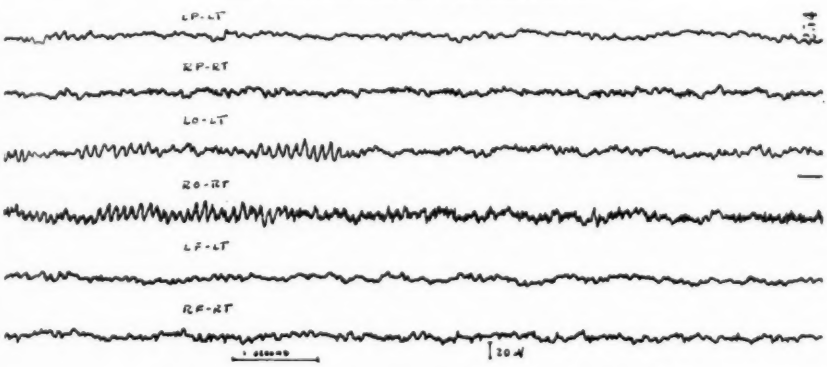

FIG. 12. Case 5. Tracing 3 weeks postoperative. The pattern is approaching what would be normal for this man.

in thickness, over the entire right hemisphere, consisting of clotted blood, was evacuated. Exploratory trephine on the left showed no evidence of extradural or subdural hematoma.

Course. Patient expired 8 hours after operation. Postmortem examination of the brain showed marked cerebral edema, uncinate hernia on the right, and laceration of the cerebral peduncle on the left by the tentorium cerebelli.

EEG. There was approximately 60 per cent decrease in amplitude over the right hemisphere with occasional low voltage 2/sec. slow waves (Fig. 13). The left hemi-

sphere showed multiple sharp 5/sec. waves and multiple 1–2/sec. high voltage waves. The tracing showed an extremely sick brain generally and was characteristic of those included in the acute phase as described by Cohn.

Case 7. C.F., a white male aged 31, was admitted on Jan. 31, 1949. He had struck his head in the right occiput 2 weeks before. He was not rendered unconscious but sought medical aid because of headache, lethargy and weakness of right side of body.

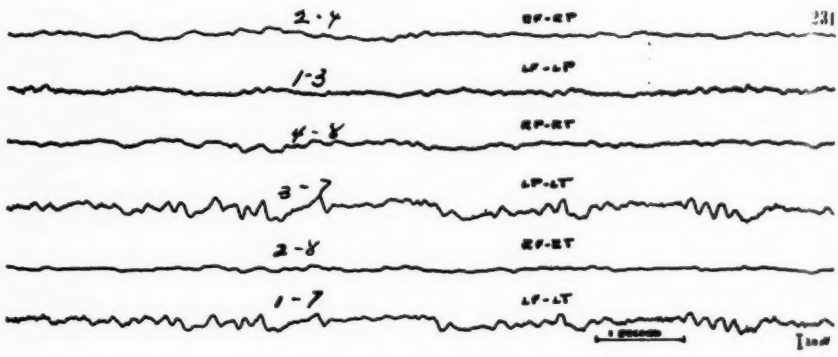

FIG. 13. Case 6. Note the extremely low amplitude over the right hemisphere, particularly over the RF-RT.

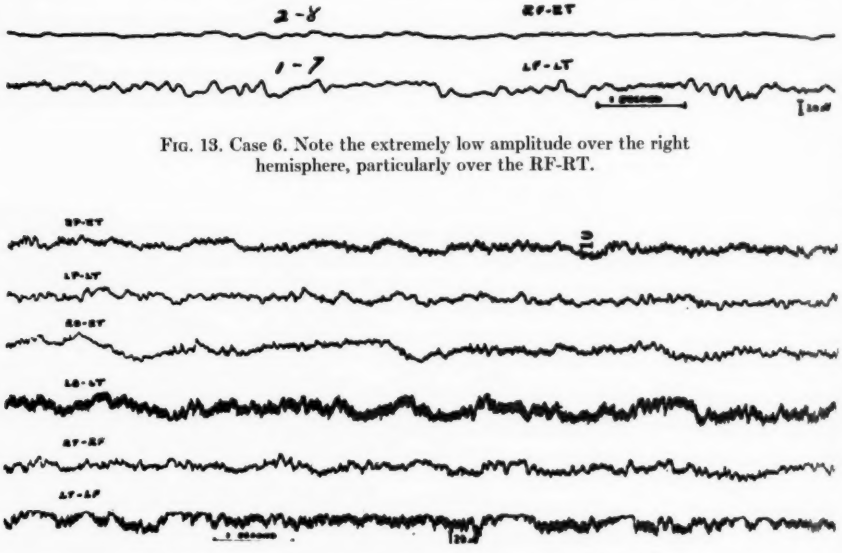

FIG. 14. Case 7. The predominant slow wave activity is over the left hemisphere with diminution of amplitude over the LP-LT areas.

Examination. Pulse was 56. He had bilateral early papilledema, right Babinski, and minimal right hemiparesis.

Operation. A left parietal trephine revealed a minimal subdural hematoma. An exploratory right parietal trephine showed no evidence of hematoma.

Course. He made an uneventful recovery and was discharged asymptomatic 17 days after operation.

EEG. The findings (Fig. 14) in this case were thought at first to be questionable or borderline for a subdural hematoma over the left hemisphere, with minimal

depression of the amplitude and multiple 2–3/sec. slow waves over the left fronto-parieto-temporal area. The fact that the layer of subdural fluid was quite thin probably accounted for the minimal EEG abnormality.

COMMENT

In 3 of the reported cases the patient showed ipsilateral hemiparesis and in 4 a contralateral dilated pupil was present. In spite of these misleading signs it was possible to predict the location of the hematoma on EEG findings. This study represents cases presented to the electroencephalographic department over a 14-month period. Of the other craniocerebral injuries that were studied with the EEG during this period, none showed electrical evidence of subdural hematoma. The accuracy of the procedure was confirmed in each case by the subsequent course of the patient.

The characteristic EEG findings in these cases were marked lowering of the amplitude and random low voltage slow waves over the affected hemisphere. This follows the pattern found in Jasper's clinical cases, and is compatible with the experimental pattern found by Cohn. There was no evidence of concurrent brain laceration or other intracranial bleeding except in Cases 6 and 7, so it was concluded that Cases 1 to 5 were uncomplicated subdural hematomas and accordingly the pre-operative tracings in these are representative of "pure" subdural hematomas. In Case 6 there was severe contusion and laceration of the brain, but the subdural hematoma was so massive that the characteristic EEG pattern was present and dominated the tracing. Case 7 had only a minimal subdural hematoma with cerebral edema and slight subarachnoid hemorrhage. Because of the paucity of subdural blood and the associated craniocerebral injuries the pattern was not as obvious or classical but suggested the diagnosis.

After a careful review of the literature on this subject it is postulated that the many conflicting EEGs taken on patients with subdural hematoma are not representative of "pure" subdural hematoma, but may well be the result of concomitant brain damage. Other observers[6,10] emphasize the frequency of the combination of vascular lesions and parenchymatous damage, or the combination of various types of hemorrhage such as subdural, extradural and intracerebral. Gurdjian and Webster,[5] in their series of 30 cases of extradural hemorrhage, stated that in 11 of these there was concomitant subdural hemorrhage. They stated "a number of patients in this series had associated severe diffuse cerebral injuries" and that they frequently found subarachnoid hemorrhage to coexist with subdural hemorrhage. It is obvious that the EEGs taken on such damaged brains will give variable findings and that the asymmetry in amplitude as described by others, and seen by us, is typical only in the "pure" subdural hematoma, and only when the bipolar leads are used over the clot.

The EEG is a diagnostic aid and is to be used only as an adjunct to the clinical findings. The EEG tracing should be correlated with the history and findings, and is in no way meant to replace careful neurological examination and observation.

CONCLUSIONS

1. The clinical course of subdural hematoma is variable and the diagnosis frequently difficult to establish.

2. A review of the literature concerning EEG in subdural hematoma revealed many conflicting reports.

3. Seven cases of surgically proven unilateral chronic subdural hematoma are presented, along with their EEG pattern.

4. With the exception of 2, these are clinically and surgically cases of "pure" chronic subdural hematomas showing no concurrent brain contusion or laceration.

5. The pre-operative EEG was characterized by a marked generalized asymmetry between hemispheres, with the hemisphere underlying the hematoma showing decreased amplitude and random low voltage slow waves. This is representative of "pure" chronic subdural hematoma tracings.

6. A period of 5 months may be required for the EEG to return to normal.

7. It is our opinion that the *variations* in the EEGs of subdural hematomas can be explained by the presence of concomitant intracranial hemorrhage and/or brain damage.

8. Failure to take a bipolar tracing with the recording electrodes over the site of the hematoma will result in an "atypical" record.

9. The EEG is of aid in establishing the diagnosis of subdural hematoma.

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LOCALIZATION OF CORD TUMORS BY ELECTROMYOGRAPHY*

PAUL F. A. HOEFER, M.D., AND SIDNEY M. COHEN, M.D.†
Department of Neurology, College of Physicians and Surgeons, Columbia University, and the Neurological Institute, New York, N.Y.

(Received for publication August 31, 1949)

NORMAL striated muscle at rest shows no sign of electrical activity.[6,10] Muscle action potentials occur as the result of all forms of physiological or pathological innervation. This is produced by impulses transmitted to the lower motor neurone from reflex fibers or by way of intermuncial neurones from descending motor tracts.[7,11,12,13] Under special conditions impulses may arise in the lower motor neurone itself.[4]

When pathological stimuli arise at segmental levels, as the result of irritative action on afferent, central or efferent structures, it is possible to utilize their effects for the localization of level lesions of the spinal cord. In case of involvement of the lower motor neurone clinical wasting of muscles supplied by affected segments is usually present to some extent. Both irritative and degenerative action may lead to visible fasicular twitches. The corresponding motor unit action potentials[4] may be recorded from inside the involved muscles by means of coaxial needle electrodes[1] and with suitable amplifiers and recording instruments.

METHOD

On this basis a method for cord tumor localization was developed several years ago.[9] It consists of systematic exploration with needle electrodes of skeletal muscles according to their segmental supply. In order to obtain simultaneous records of several muscles, ink-writing oscillographs (six-channel Grass equipment) are used. This is permissible in spite of the relative inertia of the ink-writers, because the discharge rate of single units is low (usually 6–20/sec. under the conditions of irritation or degeneration), and furthermore because only a few active units, if any, are encountered near the recording electrodes. Three or 4 needle placements in each muscle are usually sufficient to show presence or absence of spontaneous activity. In the earlier phase of this work small disc electrodes were placed on the skin over the intrinsic muscles of the hand. For technical reasons, especially precision of localization, this latter method was discontinued in favor of needle exploration of all muscles. The needle electrodes are made of hypodermic needles varying in length from $1\frac{1}{2}$ in. to $\frac{3}{8}$ in. For most purposes needles of $\frac{3}{4}$ in. length are used. The gauges of the needles vary from 27 to 25. The central wire (gauge 40 copper), the recording electrode, is insulated with baking varnish from the hypodermic needle, which serves as the indifferent electrode.

The cervical cord, including the 1st thoracic segment, is explored by recording

* Presented at the Seventy-fourth Annual Meeting of the American Neurological Association, Atlantic City, New Jersey, June 14, 1949.

† Aided by an Isador Abrahamson Fellowship from the Mount Sinai Hospital, New York City.

from the posterior neck muscles, and those of the shoulder girdle, upper arm, forearm and hand. The thoracic cord is explored by recording from the long paravertebral muscles of the back at the level of the corresponding spinous processes. Finally, muscles of thigh, leg and foot are used for exploration of lumbar and sacral segments. The method is not applied to bulbar lesions involving facial or masticatory muscles. The segmental supply of each muscle was accepted as given by standard textbooks of neuroanatomy.

In practically all instances the gain of the amplifiers is so chosen that a deflection of 10 mm. of the recording instrument corresponds to an electromotor force of 100 μV. at the input of the amplifiers. The size of motor unit discharges in man, as recorded by this and other methods, varies from about 20 μV. to almost 1 μV. The reasons for this apparent discrepancy are chiefly the unpredictable distance of the recording tip of the needle electrode from the discharging units and also actual variations in size of motor units.

Most patients tolerate easily the insertion of 6 needles with 2 or 3 changes in placement. Local anesthesia was never necessary.

Patients in whom cervical lesions are suspected are placed in a comfortable upholstered chair, reclining and with the forearm resting on the arm of the chair. By adjusting the patient's position on the chair it is usually possible to place needles at the same time in posterior shoulder girdle and upper arm muscles. In some instances, patients had to be examined on a bed, lying on one side for this procedure. Muscles of thigh and leg are explored with the patients in a reclining position on a bed. The back muscles are explored with the patient prone on the bed, usually with a pillow under the chest. Proper adjustments are made by using pillows and sandbags in order to support the limbs and to achieve comfortable positions. Incomplete relaxation and spastic postures are sources of artefact and may be misleading unless noted and corrected. Movement, another source of artefact, is easily detected. Sedation was never required. Only a small number of patients were never sufficiently relaxed to permit satisfactory recording.

Prior to an electromyographic exploration the patients are given a brief neurological examination in order to determine the approximate level involved. In other instances, where a complete hospital work-up is available, the approximate level given by the referring physician is explored. An over-all exploration of all cord levels is, of course, not feasible. The side on which exploration is started is determined by the presenting signs and symptoms. In a number of cases, evidence of a level is found more clearly or even exclusively on the opposite side. All patients suspected of thoracic lesions are bilaterally explored.

FINDINGS

The present report is based on observations of 88 cases with a total of 91 lesions, verified by operation, autopsy or unequivocally abnormal X-ray findings. The first 24 cases were the subject of an earlier report.[9]

Pertinent clinical and laboratory data of all cases were tabulated with special emphasis on levels indicated by motor and sensory findings, the clinical presence of fasciculations and on roentgenographic findings including myelography.

The tabulated material of all 88 cases in both series is statistically analyzed in the present report.

The nature of the lesions is shown in Table 1.

TABLE 1

Nature of Lesions

Fracture (and dislocation)	4
Arthritic spurs with adhesions and cord compression	7
Cervical herniation of nucleus pulposus	12
Lumbosacral herniation of nucleus pulposus	6
Lumbosacral arthritic spur	1
Schwannoma	8
Multiple neurofibromata	1
Meningioma	9
Varicosities with cord compression	2
Adhesive arachnoiditis with cord compression	4
Cord compression by tumor	1
Mediastinal tumor invading spinal canal	2
Paget's disease	1
Osteoid osteoma	1
Sarcoma	3
Multiple melanomata	1
Metastatic carcinoma	7
Dermoid cyst	2
Myelomalacia	2
Intramedullary tumor	9
Intramedullary angioma	1
Syringomyelia (and "intrinsic" cord disease)	5
Hydromyelia (in one case with platybasia)	2
Total	91

Of the 91 lesions 83 were well-defined anatomically and were considered localizable. The results of attempts at localization by electromyography are shown in Table 2. In a small number of cases, especially of thoracic lesions, a level was obtained within one segment of the operative level. This was still considered satisfactory.

In 83.1 per cent of localizable lesions electromyographic localization was thus satisfactory.

Diffuse findings over too many segments to be satisfactory, though including the proper segments, were found in 6 per cent.

A total of 8 additional lesions found to be multiple or widespread and diffuse were properly outlined by widespread electromyographic recordings.

Thus electromyographic findings gave the correct localization in 77 of 91 cases or 84.6 per cent.

TABLE 2

Localizable Lesions
Localization by Electromyography

	Correctly Localized	Diffuse Findings	False Localization	No Findings
No. of lesions	69	5	3	6
Percentage	83.1	6.0	3.6	7.2

Diffuse electromyographic findings in localizable lesions occurred in 1 instance each of:

Fracture of 5th thoracic vertebra
Schwannoma involving roots at segments C5–6
Herniation of nucleus pulposus between C4 and C5
Arachnoiditis with cord compression at 4th and 5th thoracic vertebrae
Herniation of nucleus pulposus with osteoarthritis at the interspace between C4 and C5

No electromyographic findings were obtained in 6 cases of:

1 Pancoast tumor at C8 and T1
1 instance of cord compression by thoracolumbar varicosities
3 instances of herniated nucleus pulposus at L3–4, L4–5 and L5–S1 respectively
1 instance of an osteoarthritic spur at L4-5

A majority of the negative explorations occurred thus in lesions below the extension of the cord.

In Fig. 1, 5 strips of record from a verified localization are shown. The tumor, an extradural meningioma, extended from the 3rd to the 5th cervical vertebra on the right side. The first 4 strips are tracings from trapezius, deltoid, biceps, triceps and the extensor digitorum communis on the right side. These muscles were chosen for a survey of the cervical segments. In these 4 samples, each representing 5 sec. of record, two units of different size are found discharging in the tracing from deltoid, both irregular in rate of discharge and grouping of the impulses. At the same time, a single unit is found to discharge independently in the tracing from biceps, usually at a higher rate than those in deltoid. All start and stop discharging in a characteristic abrupt fashion. No activity is noted in the tracings from trapezius and triceps. A short discharge of low grade activity is seen in one of the samples in the tracing from the extensor. In order to explore levels above C5–6 implicated by the activity found in deltoid and biceps, the supraspinatus and infraspinatus were then also investigated. The 5th strip shows a sample of this portion of the record with a continuous discharge, at the rate of 18/sec., of a single unit of infraspinatus. The findings were interpreted as suggestive of a level lesion involving the segments C4 to C6, and the findings were confirmatory as mentioned above.

Patterns of motor unit discharge found in intramedullary lesions are, in our experience, indistinguishable from those shown in the case of a meningioma.

In an attempt to appraise the relative localizing value of electromyography, these findings were compared with standard clinical methods of localization and with roentgenographic findings by flat plates and myelography in the same group of cases.

Motor level signs such as atrophy, weakness of muscles and diminution of reflexes in the localizable lesions were analyzed in identical fashion as for

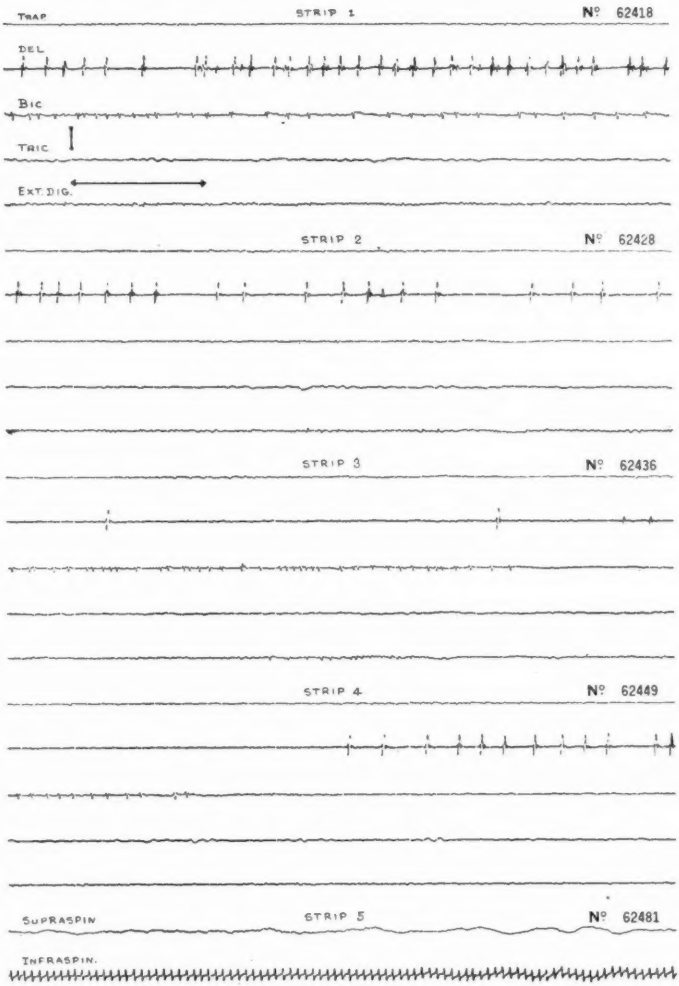

FIG. 1. Localization of extradural meningioma on the right side extending from the 3rd to the 5th cervical vertebra. The first 4 sample strips are from: trapezius, deltoid, biceps, triceps, extensor digitorum communis. The 5th strip is from supra- and infraspinatus.
Time (1 sec.) and calibration (100 μV.) are indicated on strip 1.

electromyographic level signs. The results are presented in Table 3.

This shows that a little less than 50 per cent of the lesions were established by motor signs, while in a little over 50 per cent no or misleading motor signs were found. The negative findings are only partly explained by the thoracic cord lesions.

Sensory levels are similarly analyzed in Table 4. It was interesting to note

TABLE 3

Localizable Lesions
Localization by Motor Signs

	Correctly Localized	Diffuse Findings	False Localization	No Findings
No. of lesions	41	0	13	29
Percentage	49.4	0	15.7	34.9

that, against expectation, clinical localization by sensory level was far more satisfactory than that based on motor signs.

TABLE 4

Localizable Lesions
Localization by Sensory Level

	Correctly Localized	Diffuse Findings	False Localization	No Findings
No. of lesions	56	1	15	11
Percentage	67.4	1.2	18.1	13.3

Satisfactory findings were obtained in a little over two-thirds of the cases.

Localization by flat X-ray plates in the same cases is shown in Table 5.

TABLE 5

Localizable Lesions
Localization by Flat X-ray Studies

	Correctly Localized	Diffuse Findings	False Localization	No Findings
No. of lesions	35	14	3	25
Percentage	45.5	18.2	3.9	32.4

Diffuse findings and false localizations by this method are largely due to the co-existence of osteoarthritic changes and expanding lesions, especially discs. Intrinsic cord tumors account for the negative findings.

Myelography was performed in only 40 of our cases. In these, 77.5 per cent were correctly localized, and 7.5 per cent were localized within one segment: a total of 85 per cent of satisfactory findings. Negative results were obtained in the remaining 15 per cent.

Visible fascicular twitches occurred in too few of our cases to be of localizing value.

COMMENT

Brazier, Watkins and Michelsen[2] in 1946 applied electromyography in a study of differential diagnosis of ruptured cervical disc. These authors stressed differences in their findings in cases of root compression and in

cases of degenerative processes involving anterior horn cells. We have been unable to make this differentiation except for the fact that, in most instances, the diffuse involvement of muscles in diseases such as amyotrophic lateral sclerosis or progressive muscular atrophy is borne out by electromyographic findings. On the other hand, amplitude, grouping and regularity of discharges vary in our cases from one page of the record to the next. Needless to say, a more massive degeneration of muscles will for a time lead to a larger number of units participating in the process. This, however, will change in that after the process of degeneration has run its course, the most severely involved muscles, having degenerated, will no longer show spontaneous activity of motor unit pattern, but will show fibrillation proper which is not too well recorded with ink-writing oscillographs. When that phase of the process is over, a tracing from a severely damaged muscle will be as silent as one from a relaxed normal muscle. In these situations, less severely involved muscles or muscles subject to irritation only, will show better evidence of segmental involvement than the severely damaged muscles. This is also the explanation for those cases in our series where the existence of a level was established on the uninvolved side. The size of the deflection of an action potential in a muscle depends on a variety of factors, some of which have been mentioned before, such as the actual size of the unit and the distance of the discharging units from the recording electrode. It is also conceivable that a surviving unit, in an otherwise atrophied muscle, will show less of the shunting effect of normal inactive tissue, and its discharges in this situation should show a higher electromotor force. While in a majority of our cases of cervical disc a sharp level localization was possible, several others mentioned above and 1 case of a schwannoma involving roots at C5–6 showed widespread findings, over many segments, above and below the one theoretically implicated. This is due to the compression, distortion and actual displacement of the cervical cord stressed by Bucy, Heimburger and Oberhill.[3]

Motor unit discharges may arise in motor horn cells, axones, roots and even in peripheral motor nerves in response to a sufficiently strong stimulus. The fact that fasciculations and motor unit discharges persist, in some cases, following spinal anesthesia[14,15] and sectioning of muscle nerves[5] may be explained tentatively as the result of an irritation by chemical products of degeneration.[8] As long as even a short length of axone survives, its stimulation if above threshold is bound to lead to an impulse according to the "all-or-none" law.

Irritative stimulation of reflex fibers and of internuncial neurones at the segmental level are probably the explanation for persistent fasciculation in cases without atrophy and without involvement of the anterior horn cell areas.

The fact that most of our completely negative explorations were in cases of lesions of the cauda equina and of lumbar and lumbosacral discs at L4–5 and L5–S1 would be in favor of a participation of neuronal structures above the final common path, in the production of fasciculations.

TABLE 6

Relative Localizing Value of Electromyographic Findings, Motor Level, Sensory Level and X-ray Findings (Flat Plates)

	EMG	Motor Level	Sensory Level	X-ray
Percentage	83.1	49.4	67.4	45.5

Table 6 is presented to show the relative localization value of all methods employed in our case material.

SUMMARY

Cord tumor localization by electromyography was attempted in 88 cases with 91 verified lesions.

Precise levels were established by this method in 83.1 per cent of 83 lesions considered localizable.

In 8 other lesions found at operation or autopsy to be multiple or widespread the electromyographic method had indicated the diffuse nature of these lesions.

Electromyography appears to be superior to all other methods employed for localization in our group of cases.

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MENINGIOMAS OF THE CEREBELLAR FOSSA*

ALBERT D'ERRICO, M.D.†
Dallas, Texas

(Received for publication September 8, 1949)

MENINGIOMAS of the cerebellar fossa, although few in number, deserve special study as a group. They show certain characteristic findings leading to an early diagnosis, which is particularly important for a cure. The cramped space in which they occur and their proximity to vital structures make removal hazardous, particularly since they are so vascular and so often attached to the large sinuses within the subtentorial space. Complete removal with far less hazard can therefore be accomplished before the tumor has attained too great a size.

Ten cases of meningiomas of the cerebellar fossa are presented here. These show a ratio of 1 to 5.8 in a total of 58 meningiomas and represent 2 per cent of our intracranial tumor series. In Cushing's series,[1] there were 23 cases in a total of 295 meningiomas, a ratio of 1 to 13 and representing 1.04 per cent of his total. As he noted, the incidence is much less compared to the supratentorial meningiomas than the involved dural area would indicate, for by measuring a normal adult brain he found the subtentorial dural surface to be 166 sq. cm., and the total supratentorial dural surface including the falx to be 754 sq. cm., a ratio of 1 to 4.5.

The site of attachment of these tumors in our series was found to be chiefly the tentorium and transverse and sigmoid sinuses. One of the tumors perforated the tentorium, and the ventriculograms of 1 other indicated a similar extension. The attachment of 3 situated in the angle could not be ascertained. In another in this position the attachment was to almost the entire anterior dural surface of the cerebellar fossa on one side with extension beyond the midline. In Cushing's series, similar attachments were found with specific mention of 11 tumors arising from the tentorium and the transverse and sigmoid sinuses. Of 7 tumors found in the angle an attachment was found of only 1 small one, and this was to the porus acusticus. Sufficient removal could not be accomplished in the others to ascertain the point of origin.

It appears from a study of the findings that the site of attachment determines to a large extent the symptomatology. For example, those that arose from the tentorium showed early 5th nerve changes while those situated in the angle showed symptoms characteristic of an acoustic neurinoma with early 8th nerve involvement. The most consistent and one of the earliest findings was pain in the back of the head and neck (Fig. 1). This was usually

* Presented before the Harvey Cushing Society, Hot Springs, Virginia, November 13, 1947. Case 10 added since presentation.

† 1431 Medical Arts Building, Dallas 1, Texas.

of long standing, and in Case 7 was present for 17 years. For the most part, it was severe and was usually more marked on the affected side and increased by bending the neck forward or turning the head to either side.

Nausea alone was present in 2 cases and was associated with vomiting in 6. These symptoms occurred either early or late in the course of the illness and usually accompanied spells of headache or dizziness. Ataxia of the extremities or staggering gait was present in all but 1 case, and this was usually

Patients	Headache Neck Pain	Nausea Vomiting	V th	VIII th	Weakness	Hypesthesia	Position Sense Astereognosis	Ataxia	Dysphagia
(1) Jones (Left)	Pain back of head and neck	Vomiting late	—	—	Weakness Rt. leg	—	—	Staggering	—
(2) DeWitt (Left)	Pain back of neck	Vomiting late	Hypesthesia Left side of face	Deafness Left Early	—	—	Astereognosis Left hand	Staggering	In spells
(3) Wilson (Left)	Pain back of head and neck	—	Hypesthesia Rt. side of face	Deafness Left	—	Rt. arm	—	—	—
(4) Grissom (Right)	Pain back of head and neck	Nausea	—	—	—	Left arm and leg	—	Staggering	—
(5) Edwards (Left)	Pounding pain back of neck	Nausea	Hypesthesia Rt. side of face	Vertigo Early	Weakness Left arm and leg	—	Impaired position sense Left arm and leg	Falls to left. Ataxia left finger to nose	Late
(6) Eikel (Left)	Headache, stiffness, pain back of neck	Nausea, vomiting early	—	—	—	—	Impaired position sense Left arm	Staggering, ataxia left finger to nose	
(7) Miller (Right)	Pain back of head and neck (17 years)	Vomiting early	—	Vertigo Deafness Rt. since childhood	Weakness Rt. arm	Rt. arm and leg	Astereognosis Rt. hand	Staggering, falls to right	Late
(8) Rogers (Left)	Headache stiffness back of neck	Vomiting late	Hypesthesia Rt. side of face	Deafness Rt. 1 yr.	—	—	—	Ataxia Finger to nose Rt. and left	—
(9) Thomas (Right)	Pain rt. side head, back of neck	—	Hypesthesia right side of face	Deafness right 1 yr.	—	—	Impaired position sense rt. arm and leg	Staggering, ataxia rt. finger to nose	Late
(10) Hollis (Right)	Headache, stiffness back of neck.	Nausea, Vomiting	Hypesthesia Rt. side of face	Deafness Right	—	—	Impaired position sense right arm	Ataxia rt. finger to nose	Late

FIG. 1. Table showing prominent symptoms in all 10 cases of meningiomas of the cerebellar fossa.

a comparatively early symptom. Hypesthesia of the face, at times associated with tingling, was remarkably common, being present in 6 cases. It is interesting to note that this occurred on the same side as the lesion in 3 instances and on the opposite side in 3. The 8th nerve was involved in 7 cases, with deafness alone in 5, vertigo alone in 1, and vertigo together with deafness in Case 7. The deafness was on the same side as the lesion in 5 cases and on the opposite side in Case 8. Definite facial weakness was seen in only 2 cases.

Tract signs were quite common. Weakness of the extremity was present

in 3 cases, involving the contralateral leg in one, the ipsilateral arm in another, and the ipsilateral arm and leg in a third. Hypesthesia was found in the extremities of the same side in 1 case and of the contralateral side in 2. Neither astereognosis nor loss of position sense was found to be contralateral in any case, ipsilateral astereognosis being present in 2 cases and ipsilateral loss of position sense in 4. All of these tract signs were usually fairly late in occurrence. Dysphagia was present in 5 cases, occurring late in the illness. Papilledema was found in 4 cases when first seen.

The subtentorial meningioma is most commonly confused with an acoustic neurinoma, and when the 8th nerve is involved late or not at all the symptoms often are attributed to some other cause. The common occurrence of 5th nerve involvement has already been noted, the hypesthesia having been on the opposite side of the lesion in 3 of the 6 cases in which it was present. This hypesthesia usually began early in the history, probably due to the high location of the tumors. With acoustic nerve tumors, the hypesthesia comes on later and is only rarely found on the opposite side of the lesion. These characteristics are helpful in the differential diagnosis. Involvement of the side opposite to that expected was also seen with the tract signs of weakness and hypesthesia. Such evidences of bilateral involvement might be considered to be due to a tumor within the pons and medulla, although undoubtedly they are due to transmitted pressure. Tract signs were present in every case except Case 8, thus being much more common than with the acoustic neurinomas. Nausea and/or vomiting were present in all but 2 cases; dif-

SPINAL FLUID PROTEINS

ACOUSTIC NEURINOMAS			MENINGIOMAS		
Case	Name	Spinal Fluid Protein mg.	Case	Name	Spinal Fluid Protein mg.
6	Davis	280	2	DeWitt	65
10	Morrow	300	3	Wilson	20
11	Dedacheck	85	5	Edwards	15
15	Tweedy	725	6	Eikel	50
17	Wood	120	7	Miller	5
19	Fite	100+	9	Thomas	40
23	Sefcik	195			
24	Jones	230			
27	Dollins	50			
AVERAGE PROTEIN		231 mg.	AVERAGE PROTEIN		35 mg.

FIG. 2. Table showing spinal fluid proteins of acoustic neurinomas compared with those of meningiomas of the cerebellar fossa.

ficulty in swallowing was present in 5; and only 1 showed no evidence of ataxia. All of these symptoms were in higher proportion than is found with the acoustic neurinomas.

One of the most important differential findings is the spinal fluid protein. As shown by de Vet,[2] the protein is markedly elevated in practically every case of acoustic neurinoma. In our tumor series, spinal fluid examination was done in 9 cases of acoustic neurinomas and 6 of meningiomas of the cerebellar fossa. It will be seen in Fig. 2 that there is a striking difference in the spinal fluid proteins, with an average of 231 mg. for the acoustic neurinomas and 35 mg. for the meningiomas.

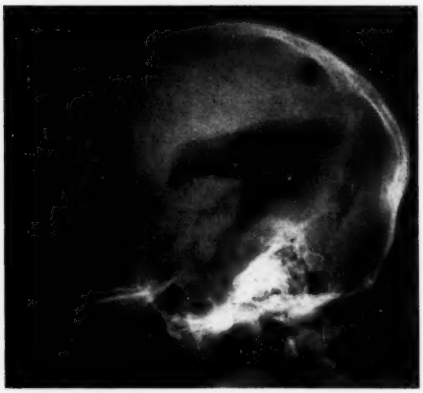

FIG. 3. Ventriculogram showing both lateral ventricles with characteristic doming of under border of ventricle on side of lesion compared with normal contour on opposite side.

Ventriculograms were done in 4 cases of acoustic neurinomas and 5 of meningiomas of the cerebellar fossa. While this number is admittedly small, comparison shows little or no change in the contour of the temporal or occipital horns with the acoustic neurinomas but a significant and characteristic change in almost every case of the meningiomas. This change is usually a shallow doming of the under border of the temporal and occipital horns on the side of the lesion, but where there is extension of the tumor through the tentorium the defect is more circumscribed and deep (Figs. 3 and 4). The changes usually show best in the lateral view. This finding, therefore, appears

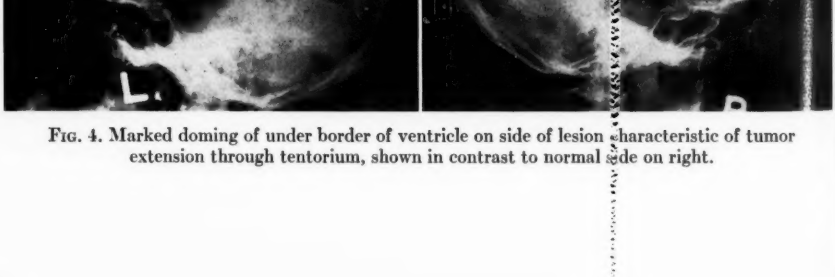

FIG. 4. Marked doming of under border of ventricle on side of lesion characteristic of tumor extension through tentorium, shown in contrast to normal side on right.

to be characteristic of a meningioma of the cerebellar fossa and is of definite value in lateralizing the lesion. With the conflicting neurological signs this is particularly important, since a unilateral approach is preferable for complete removal.

The approach best suited to removal of these meningiomas is through a unilateral combined occipital and suboccipital flap. With this procedure, a large unilateral skin and muscle flap is elevated from the bone over the occipital and suboccipital regions on the side of the lesion. Four burr holes are made, two well below the transverse sinus and two at least 4 cm. above. The two medial openings are just lateral to the midline and the lateral openings are in line with the inner border of the mastoid process. All are connected with a Gigli saw and the bone removed is replaced at the time of closure. An adequate occipital portion of the flap is one of the most important features in the operation, for adequate exposure of the tentorium is thus obtained, and the advantage of this is obvious since so many of the tumors arise from the tentorium and lateral and sigmoid sinuses. The outer third of the cerebellum is resected up to the tentorium, and it may be necessary to carry this removal more medially above the dentate nucleus for sufficient exposure. When these tumors are large, the small space in which they occur necessitates either preliminary piecemeal excavation or the removal of a large wedge. Bleeding was much more profuse in these lesions than in the supratentorial ones, so that multiple stages had been the rule. With large tumors, removal even to the attached stalk has seldom been possible heretofore, but with the added approach obtained by bone removal above the tentorium, extirpation is greatly facilitated. With this exposure, complete removal with the attached tentorium and sinus was effected in Case 4. The complete specimen weighed 97 gm. (Fig. 5). The patient stood the procedure well and showed no untoward effects other than an accumulation of fluid beneath the muscles which necessitated aspiration every day or two over a period of about 2 weeks. There were 2 postoperative deaths in the series, 1 after the 1st stage and 1 after the 4th stage. These were the largest tumors of the group.

FIG. 5. Case 8. Specimen removed completely with attached tentorium and transverse sinus; weight 97 gm.

The histology of these tumors shows a high proportion of the fibrous and meningothelial types. Only 2 psammomas were in the series.

SUMMARY

Ten cases of meningioma of the cerebellar fossa are reported. With an analysis of the findings in this group of patients, certain characteristic features were found which could have led to a much earlier diagnosis when the tumor would have been smaller and more amenable to complete removal. The histories were usually long, and in Case 7 it was 17 years. These lesions are slow in growth, and even with incomplete removal the patients have long survival periods. In this series, other than for the 2 postoperative fatalities, there has been 1 death 3½ years after the 2nd stage. Although complete extirpation of large tumors even with invasion of the lateral sinus or tentorium can be accomplished, diagnosis at an early stage is important since a complete removal should then be the rule, with little if any disability remaining.

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TRANSABDOMINAL EXTRAPERITONEAL SECTION OF THE OBTURATOR NERVE TRUNK

PAUL H. HARMON, M.D.

Department of Orthopedic Surgery, Permanente Hospitals and The Permanente Foundation, Oakland, California

(Received for publication September 8, 1949)

A POPULAR method of interrupting section of the obturator nerve is to section its many peripheral branches high in the medial thigh as originally described by Stoffel[6,7] in 1910. However, obturator nerve section in the thigh is frequently not as effective as section of the trunk higher because of accessory obturator nerves and branches of the main obturator trunk which may originate within the abdomen and pursue a variable peripheral course.

Selig[4,5] in 1913 and 1914 reported an anatomical study demonstrating the possibility of low intrapelvic extraperitoneal section of the obturator trunk. A number of authors (reviewed by Chandler and Seidler[2] and by Wischnewsky[8]) have reported on the use of this technique. Chandler and Seidler[2] reported 84 cases in 1939, in which the nerve was approached through a lower abdominal incision, just lateral to the lower border of the rectus muscle. In cases of bilateral section of the nerve these authors made a transverse skin incision with vertical deep dissection on the lateral side of each rectus abdominis muscle. Bonnet[1] described a lateral iliolumbar approach through which the obturator nerve was located high beneath the iliopsoas muscle. The disadvantage of this technique is the lengthy incision and deep dissection. Recently, Freeman[3] reported the combined section of the obturator and femoral nerves in paraplegics, through a single vertical incision which crossed Poupart's ligament. The approach to the obturator nerve, as described by this author, was essentially the Selig-Chandler approach. The femoral nerve was sectioned below Poupart's ligament.

During the past two years, we have employed a convenient transabdominal extraperitoneal approach to the obturator nerve high and laterally within the pelvis at the level of the anterior superior iliac spine. The details of surgical technique are described below and illustrated in Fig. 1.

The advantages of this operation are:

(1) The nerve trunk is sectioned high enough to include all the branches.

(2) The nerve is easily located in a field where it is unlikely to be confused with other structures.

(3) The operation has the advantage of all extraperitoneal approaches in being bloodless and non-shocking.

(4) The femoral and other nerves of the lumbosacral plexus can be sectioned either through the same approach or an upward extension of it.

(5) The anatomic repair of the muscle-splitting incision allows the

patient to rise the same day of operation without danger of postoperative hernia.

We have performed 23 bilateral sections of the obturator nerve and 3 unilateral sections with no complications. Sixteen of the bilateral nerve sections have been in paraplegics in whom section of the femoral nerve was also performed through the same muscle-splitting incision. In these cases the femoral nerve was located in its downward course between the iliac and psoas divisions of the iliopsoas muscle, also at the level of the anterior superior spine. One small incision and one dissection suffice for the section of these two nerves on a single side.

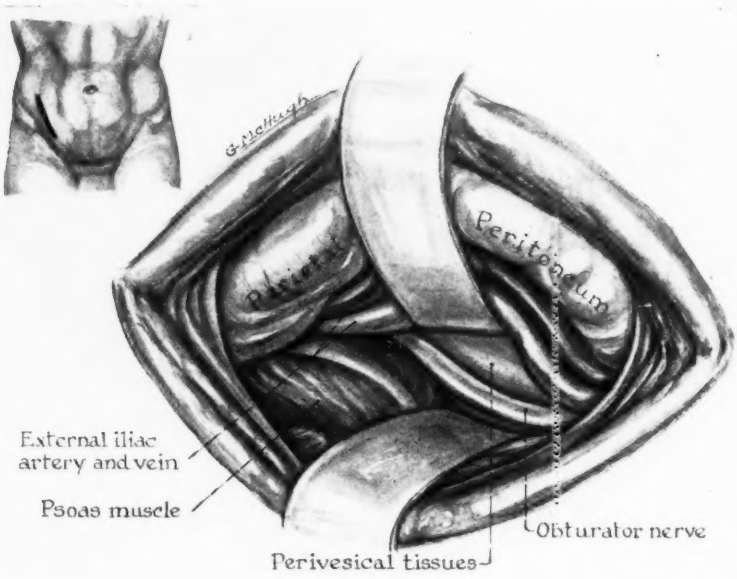

FIG. 1

OPERATIVE TECHNIQUE

When both obturator nerves are sectioned, the skin is divided by a transverse lower abdominal incision. When a single nerve is sectioned, a slightly oblique 4 in. lower right or left quadrant skin incision is made. The abdominal muscles are then split in the direction of their fibers, as in the classical McBurney incision. As the preperitoneal fat is reached, the peritoneum is separated without opening and is pulled medially by stick-sponge dissection at the level of the anterior superior iliac spine. Posterior and medial dissection along the anterior surface of the iliacus fascia in the internal iliac fossa at the same level exposes the external iliac artery at the pelvic brim. The artery and the accompanying vein are carefully freed from the

lateral wall of the pelvis by blunt dissection. The external iliac artery and vein are then retracted medially in order to give a 1 in. wide interval between the lateral pelvic wall and the vessels. Deeper dissection in this opening reveals the obturator nerve slightly below and slightly lateral to the retracted external iliac vein, which is the deeper of these vessels.

The obturator nerve in its upper and middle course in the pelvis is a free-lying trunk, 1 to 3 mm. in diameter. There is no other structure in this locality with which it can be confused. The nerve is elevated by a blunt hook. During the process of elevation the nerve is usually irritated enough so that it is also at the same time identified by the resulting irregular stimulation of the adductors of the corresponding leg. The trunk is then sectioned with a cautery. The central end is turned upward, the retractors are removed, and the structures allowed to resume their normal relations. It is then necessary to perform only a layer closure of the abdominal wall without drainage. The patient rises on the same day of the operation and usually leaves the hospital the next day. The structures as viewed by the operator just before section of the nerve are shown in Fig. 1.

The only difficulty encountered by us has been in 2 cases in paraplegics, who also had a thickened bladder and pericystic inflammation due to urinary tract infection. In these cases the tissues in the lateral pelvis were indurated and adherent and the dissection deep to the external iliac arteries was slightly more difficult. In 1 of these cases the obturator nerves were embedded in scar, apparently produced by the same inflammatory process.

SUMMARY AND CONCLUSIONS

A convenient technique for abdominal extraperitoneal section of the obturator nerve at the level of the anterior superior iliac spine is described. This approach is especially useful for combined obturator and femoral nerve section in paraplegics who require relief from involuntary flexion-adduction hypertonus.

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VENTRICULOMASTOIDOSTOMY

TECHNIQUE AND OBSERVATIONS

WILLIAM A. NOSIK, M.D.*
Cleveland, Ohio

(Received for publication September 30, 1949)

IN IDIOPATHIC congenital communicating hydrocephalus or in the progressive hydrocephalus secondary to a purulent meningitis, the existing imbalance between the production and absorption of the cerebrospinal fluid may impose an almost insurmountable problem in the control of the intracranial pressure. In an historical review of the subject one is impressed with the number and variety of surgical schemes that have been proposed to correct this unhappy clinical situation. Though, in some instances, great ingenuity has been demonstrated in the effort to solve the problem, each newly proposed surgical measure has met with a degree of failure unique unto itself. To this imposing list I should like to add another procedure which, in spite of its fault, has been effectively used as a palliative measure—the ventriculomastoidostomy, wherein the ultimate step of external drainage of cerebrospinal fluid has been taken.

In this procedure, first performed on March 28, 1947, an attempt is made to establish a pathway from the lateral ventricle into the antrum of the mastoid through a polyethylene tube to which a special tantalum tip has been affixed. The fluid passes from the ventricle into the antrum of the mastoid, through the atrium and then drains via the Eustachian tube into the nasopharynx (Fig. 1 C).

While the thought of an open pathway from the ventricle to the nasopharynx is abhorrent to neurosurgeons, it is believed that the presence of a constant higher intracranial pressure as well as the normal movement of the Eustachian tube cilia results in an undirectional flow of fluid in an outward direction to decrease the risk of retrograde infection. A limited clinical experience appears to bear out this statement.

Because of its simplicity this operation may have some interest in a clinical application. There is presented here an outline of the surgical technique.

TECHNIQUE

1. In the selected patient x-ray study is made to determine the degree of pneumatization of the mastoid cells, which if inadequate will preclude the establishment of a free channel.

2. The ear is checked to eliminate the possibility of infection.

3. The ear and temporomastoid area (usually right side) are prepared for surgery, and under general anesthesia a curvilinear incision, 6–7 cm. long, is made above and behind the ear (Fig. 1 A).

* 10515 Carnegie Avenue, Cleveland 6, Ohio.

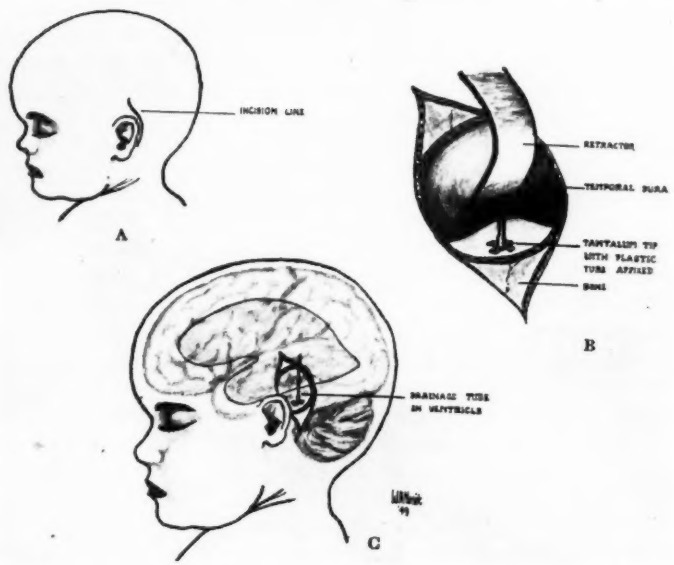

FIG. 1. (A) Placement of incision. (B) Appearance of operative area with plastic tube and tantalum tip in place. (C) Schematic drawing demonstrating relations of drainage tube and ventricle.

4. A trephine opening is made through the temporal bone to expose the posterior portion of the middle fossa. The mastoid is not opened.

5. The dura is stripped from the petrous portion of the temporal bone on the dorsal surface to a point medial to the superior arcuate eminence.

6. To locate the antrum of the mastoid a bright Cameron light is introduced into the ear canal. The antrum transilluminates with greater intensity than does the surrounding structure.

7. The antrum is punctured with a dental probe and the edges of the punctate opening cauterized carefully. Any residual blood in the antrum cell must be carefully removed. The dura directly over the antral puncture is then cauterized at this point and a small opening made to admit the plastic tubing.

FIG. 2. Polyethylene tube with tantalum tip affixed.

8. A piece of polyethylene tubing, 5–6 cm. long, having an inside diameter of 1 mm., is tightly affixed to a prepared tantalum tip (Fig. 2). A curved stylet is introduced into the tube and the tube inserted into the temporal horn of the lateral ventricle, the stylet then being withdrawn. A free flow of clear fluid must be obtained.

9. The tantalum tip is then inserted into the previously made puncture-hole in the antrum and tightly anchored by pressing the prongs into the petrous bone. Gelfoam pads may be placed about the tip and the brain re-expanded with saline solution (Fig. 1 B).

10. The disc of bone is replaced and the incision closed in the usual fashion.

11. One cc. of indigo-carmine may be injected into the ventricle to determine the patency of the drainage pathway postoperatively. Penicillin and/or sulphonamides have been used recently in the first month after operation.

OBSERVATIONS

In the past 3 years 9 patients whose clinical condition was regarded as poor have been subjected to this procedure. In this series of 9 patients, meningitis developed in only 1, and that 1 month postoperatively. No organism was cultured but the cellular response in the cerebrospinal fluid plus mild clinical signs made this diagnosis mandatory. It should be stated, however, that a free drainage was not satisfactorily established. The longest postoperative survival to date is 2 years and 9 months, and, in spite of the patient having had measles as well as several severe upper respiratory infections, no retrograde infection followed. This patient is still draining clear cerebrospinal fluid in small quantities when she cries. The head circumference at 7 months was $21\frac{3}{8}$ in.; the chest $16\frac{1}{8}$ in.; at 2 years and 9 months head circumference was 22 in. and the chest $20\frac{3}{4}$ in. The patient was alert and was showing definite progress in learning to speak, stand and walk.

One patient died $18\frac{1}{2}$ months after operation; ventriculomastoidostomy for idiopathic congenital communicating hydrocephalus had been done at age of $4\frac{1}{2}$ months. The head circumference was $18\frac{7}{8}$ in. preoperatively and 21 in. before her death. The postmortem findings referable to the brain were:

Gross Examination. The convolutions were flattened, with narrow sulci. A functioning ventriculomastoidostomy was present. Serial sections of the brain showed marked dilatation of the ventricular system. The cerebral substance was markedly thin and compressed, measuring only 6 mm. in some areas. The internal capsule was displaced and distorted.

Microscopic Examination. Vascular engorgement with marked dilatation of the Virchow-Robin spaces was present. There was distortion of nerve cells and fibers in all areas of thin cerebral substance. In the lateral ventricles ependymal cells were absent, exposing poorly cellular glial tissue which formed a smooth lining. The ependymal lining of the 3rd and 4th ventricles was well preserved in numerous small areas which were separated by prominent bulbous intrusions of glial tissue. A fine papillary appearance resulted in some areas.

Repeated skull films taken postoperatively in these patients showed no evidence of intracranial air. When the tube is functioning well and a satisfactory drainage has been established, an injection of indigo-carmine into the lateral ventricle results in a colored drainage from the Eustachian tube within several minutes. If at the time of the injection the ear drum is viewed with an otoscope, a bluish discoloration in the fluid can be seen advancing across the drum until the middle ear has filled with the colored fluid.

In the first operation, a tube of 2 mm. inside diameter was used. It was believed that too rapid a decompression followed and may have been responsible for the patient's death 12 hours postoperatively with a hyperthermia. Since then a tube of 1 mm. diameter has been used.

Because there is no fluid loss from the body no new problem is introduced. Where this procedure was applied in an adolescent there was no complaint referrable to the ear on the operative side at any time.

This operation has been performed in infants with idiopathic congenital communicating or non-communicating hydrocephalus, and in several other older patients having intracranial malignancy with obstruction of the aqueduct. If a lesion obstructs the interventricular communication the unilateral procedure obviously would be unsatisfactory.

Where the patient's condition did not permit a long operation, designed either to re-route the cerebrospinal fluid around an obstruction or to decrease the amount of choroid plexus present, the ventriculomastoidostomy was well tolerated.

The tantalum tip may be obtained from the Ethicon Suture Laboratories, New Brunswick, New Jersey; the polyethylene tubing from Surprenant Mfg. Company, 199 Washington Street, Boston 7, Massachusetts.

THE RESULTS OF 300 PITUITARY ADENOMA OPERATIONS (PROF. HERBERT OLIVECRONA'S SERIES)

LOUIS BAKAY, M.D.*

Neurosurgical Clinic, Serafimerlasarettet, Stockholm, Sweden

(Received for publication October 11, 1949)

BETWEEN the years 1929 and 1947, 292 patients with verified pituitary adenoma were treated at the Serafimerlasarettet in Stockholm and the number of operations performed was 300. The detailed follow-up of almost all patients allows an accurate analysis of the value of these operations and of the postoperative prognosis. There is only one similar follow-up study in the medical literature, Harvey Cushing's 338 pituitary adenoma cases as reported by Henderson[7] in 1939.

The 292 cases of pituitary adenoma observed in the Olivecrona series represented 8.9 per cent of the total number of verified brain tumors. On the other hand, the 338 corresponding cases in Cushing's series represented 17.8 per cent of a total of 2023 brain tumors—a larger percentage.

Histologically, 232 of the adenomata have been classified as chromophobe (with 246 operations); 55 as acidophil or mixed but with an overwhelming majority of the acidophil elements (52 op.); 2 as basophil (2 op.); and 3 as malignant (3 op.).

The maximum incidence of chromophobe adenomata occurred in patients between the ages of 50–54, much higher than in Cushing's series. Twenty-six patients were over 60 years of age. The age incidence of patients with acidophil tumors was lower, about 30 years of age, and only 1 of them was over 60.

In 55 (25.9 per cent) of the cases of chromophobe adenoma and in 18 (35.3 per cent) of the cases of acidophil adenoma, the patients had received varying periods of x-ray treatment during the years or months prior to operation.

The types of operations performed are recorded in Table 1. This table does not include 13 re-explorations performed shortly after primary operation for actual or suspected blood clot formation or edema of the frontal lobe. The re-explorations were followed by resection of the frontal lobe in 4 cases, a rare procedure in the primary operations. Primarily it was performed in only 4 cases, in which the approach to the big extrasellar adenomata was not possible by other means.

The surgical approach was transfrontal in 241 chromophobe and 45 acidophil adenoma cases (95.3 per cent of all operations), and transsphenoidal in 5 chromophobe and 9 acidophil cases (4.7 per cent). A fundamental difference exists, therefore, between our cases and Cushing's statistics, the transfrontal

* Present address: Department of Neurosurgery, Massachusetts General Hospital, Boston 14, Massachusetts.

technic having been used in almost all of our pituitary operations, whereas most of Cushing's patients were operated upon transsphenoidally (64.6 per cent of the chromophobe and 90 per cent of the acidophil adenomata).

There were 28 (11.3 per cent) postoperative fatalities after 246 chromophobe adenoma operations, 4 (9.5 per cent) after 52 acidophil, 2 after 3 malignant, and none after the 2 basophil operations. The results improved with time, however, and the series of 30 acidophil adenomata operated upon in the last 10 years had the remarkably low mortality rate of 3.3 per cent. All patients who remained in the hospital and died there from any cause are

TABLE 1

Types of Operations Performed

	Chromophobe	Acidophil	Basophil	Malignant
Intracapsular enucleation	232 (ex. 22)	45 (3)	2 (0)	2 (1)
(a) transfrontal	229 (22)	35 (2)	2 (0)	2 (1)
(b) transsphenoidal	5 (0)	10 (1)	—	—
Partial extirpation	10 (6)	1 (0)	—	1 (1)
Biopsy	3 (0)	—	—	—
Exploration	1 (0)	2 (1)	—	—
Subtemporal decompression	—	1 (0)	—	—

included in these figures. The postoperative mortality rates of Cushing's 338 cases were 4.9 per cent for the chromophobe and 8.6 per cent for the acidophil, without any essential difference between the transsphenoidal and the transfrontal operations. Grant's[5] series (143 pituitary adenomata) showed a mortality rate of 11.3 per cent for the transsphenoidal and 9.5 per cent for the transfrontal route. It is obvious that there are fewer postoperative deaths in cases of small intrasellar growths and that the mortality rate is much higher when there is extrasellar extension. Jefferson[8] reports 2 per cent mortality in the first and 33 per cent in the second group. In our series there were 14 fatalities (35 per cent) after operations for chromophobe adenomata with extension (40 cases), and 14 deaths (6.4 per cent) after operations for tumors without extension.

The causes of deaths are listed in Table 2.

Most fatalities are due to the incomplete removal of large adenomata invading the hypothalamus and the 3rd ventricle. The extirpation, however, of such tumors is hardly possible. Hyperthermia without any other symptom followed by death has been observed in 3 cases. Hyperthermia is a well known factor in pituitary operation statistics and is supposed to be the sign of a hypothalamic crisis, although its origin is not clear.

EXTRASELLAR EXTENSIONS

It is known that a certain number of pituitary adenomata tend to extend into the intracranial chamber. According to the estimation of Henderson[7]

and Jefferson,[8] the incidence of extrasellar extension is about 14 per cent.

Extension in the *cavernous sinus* was present in 15 chromophobe and 1 malignant adenomata (6.8 per cent). In these cases the laterally growing neoplasm maintains a permanent and increasing pressure on the medial wall of the sinus or invades it and injures the structures that it contains, particularly the oculomotor nerve, as described by Boschi and Campailla,[1] Weinberger, Adler and Grant,[10] and Jefferson.[8] The first symptom is usually a slight disturbance in coordination of the ocular muscles and diplopia (2 cases) due to weakness of the 3rd nerve. Palsy of the nerves leading to the muscles of the eye occurs in more advanced cases and varies largely in ex-

TABLE 2

Causes of Death

	Chromophobe	Acidophil	Malignant
Partial removal, followed by increased intracranial pressure	9	3	1
Pituitary insufficiency	4	—	—
Postoperative clot formation	2	1	—
Hemorrhage into ventricles	1	—	—
Thrombosis of anterior cerebral artery	—	—	1
Hyperthermia (hypothalamic crisis)	2	1	—
Meningitis	1	1	—
Pneumonia	2	—	—
Cardiac insufficiency	1	—	—
Pulmonary embolism	1	1	—
Miliary tuberculosis	1	—	—
Scarlet fever, endocarditis	1	—	—

tension and severity. In our series partial or complete oculomotor nerve palsy was observed in 11 cases, paralysis of the trochlear nerve in 2, and paralysis of the abducens nerve in 2. It is easy to understand that the extension of an adenoma in the cavernous sinus is a sign of asymmetrical growth and therefore causes homonymous hemianopia or only one-sided temporal hemianopia more frequently than the usual type of adenoma. The lateral extension does not always mean that the neoplasm has also extended upwards; a lesion of the nerves of the cavernous sinus, without injury of the optic nerve, has been observed in some cases.

Protrusion of the eyeball on one side, as occurred in 2 of our patients, is also caused by the invasion and the resulting venous congestion of the cavernous sinus. It is diminished after removal of the tumor.

Jefferson thinks that the existence of symptoms originating in the structures of the cavernous sinus does not mean a poor prognosis unconditionally but he classified the invading type of these adenomata as malignant. In our cases, however, there was only 1 that could be classified as malignant by the microscopic picture; it resulted in total 3rd and 4th nerve palsy on the left side. It is remarkable that 3 of the 16 patients have been operated upon later for recurrence of symptoms, a moderately high rate of recurrence, but

on the other hand, it is difficult to decide if this was due to the malignancy of these neoplasms or only to the fact that it is extremely difficult to remove an adenoma completely from the wall of the cavernous sinus. The number of postoperative fatalities in this group with extension into the cavernous sinus was not higher than with the usual adenoma (2).

Obstruction of the *internal carotid artery* can be caused also by extension in the lateral direction, as observed in 2 of the chromophobe cases. In 1 the neoplasm grew upwards, following the path of the artery. In the other the adenoma surrounded the left carotid artery and obstructed it, later causing disturbances in cerebral circulation, slight hemiparesis on the right side, facial weakness, slight aphasia and agraphia.

Another sign of lateral extension is invasion into the *cave of Meckel.* Trigeminal pain or anesthesia due to this extension (described by Jefferson as a sure sign of malignancy) was observed in 3 cases, 2 chromophobe and 1 acidophil. In 1 case autopsy revealed a large tumor growing upwards into the 3rd ventricle and extending widely on the base of the skull, infiltrating the semilunar ganglion.

One example of marked *temporal extension* can be presented in our series. The extension to the right side was shown by atrophy of the right optic nerve with total blindness, while the left optic nerve and vision were normal with but a slight temporal shrinking of the visual field. There were, in addition, paralyses of the right oculomotor and hypoglossal nerves, and a slight hemiparesis on the left side.

Although there were several adenomata among the chromophobe and malignant cases that showed infiltration into the sellar floor, erosion of the base of the skull and extension into the *nasopharynx* could not be observed. Another rare pathway of extension is backwards and downwards, the adenoma growing *below the tentorium* (White and Warren[12]). In 1 of our cases the occipitally growing tumor compressed the cerebral peduncles and injured the pyramidal tracts.

The projection of the adenoma upwards into the 3rd ventricle, the so-called *hypothalamic extension*, is of great prognostic importance. Compression of the hypothalamus and invasion of the 3rd ventricle frequently make the outcome of operation unfavorable. The most striking examples were represented by those 14 adenomata (11 chromophobe, 2 acidophil and 1 malignant, altogether 4.7 per cent) that protruded into the 3rd ventricle and filled it almost completely, blocking the circulatory pathway of the cerebrospinal fluid, with resultant hydrocephalus and symptoms of increased intracranial pressure. Some of the rare cases of papilledema have been found in connection with these neoplasms. In 1 of our patients the tumor did not remain localized to the 3rd ventricle but extended upwards in the right lateral ventricle through the interventricular foramen. The postoperative mortality rate of this group was high (71.4 per cent).

In less advanced cases hypothalamic symptoms were caused by compression of this region. Polydipsia and polyuria (alteration of the supraoptic

nucleus and its connections) were present in 20 cases of chromophobe tumors (8.5 per cent) and drowsiness occurred in 14, the latter originating probably in injury of the hypothalamic sleep-centers (Jefferson).[8] There are three different periods in pituitary adenomata in which hypothalamic symptoms can develop. A preoperative lesion is due to direct pressure on the hypothalamic nuclei by the upwardly extending tumor; the lesion diminished or disappeared after removal of the adenoma with but 1 exception. Another type of disturbance occurs in the early postoperative period, lasts some days or weeks and is usually transitory. Good effect is to be expected from treatment with posterior lobe extracts. In this period the symptoms are caused probably by surgical manipulation and injuries of the hypothalamus. The third group consists of cases in which the symptoms start years after the operation and suggest a recurrence of the adenoma. The mortality rate of patients with primary polydipsia and polyuria is high and the late results of operation are not favorable. Four of the patients died following enucleation of the tumor, and 5 others within 5 years, altogether 45 per cent of this group. Transitory postoperative polydipsia and polyuria were observed in 6 cases, but in none of these became permanent.

Polydipsia and polyuria were found in 12 cases (22.7 per cent) of acidophil adenoma. There were 3 postoperative fatalities, and 2 other patients died within the next 2 years (together 41.6 per cent). Postoperative transitory polydipsia developed in 1 patient; the absence of this complication in the others can be explained by the fact that several patients were operated upon through the transsphenoidal approach, a method that certainly does not injure the hypothalamus.

Two patients with chromophobe adenoma suffered from epileptic seizures and 3 showed mental disturbances. These signs are not frequent in connection with adenomata of the pituitary body, but, if present, are a danger signal indicating that the tumor is expanding widely beyond the sella turcica (Jefferson;[8] White, Liu, and Mixter[11]).

The estimation of the size and location of the extrasellar extension makes the operative planning easier. Encephalography, especially the filling with air of the basal cisterns, is of great help (Figs. 1, and 2, and 3).

OCULAR SYMPTOMS

Visual Acuity

Chromophobe Adenomata. The preoperative examinations showed that the vision of 159 patients (68.5 per cent) was affected in both eyes. Unilateral blindness was present in 17 cases and bilateral in 1. The operative mortality rate of this group was 10.7 per cent (17). The vision of one eye only was diminished in 63 cases (27.2 per cent); in 8 cases there was blindness of one eye. The mortality of this group increased to 17.4 per cent (11). Finally, normal vision of both eyes was recorded in 10 patients (4.3 per cent); none of them died following operation. The small number of this last category in-

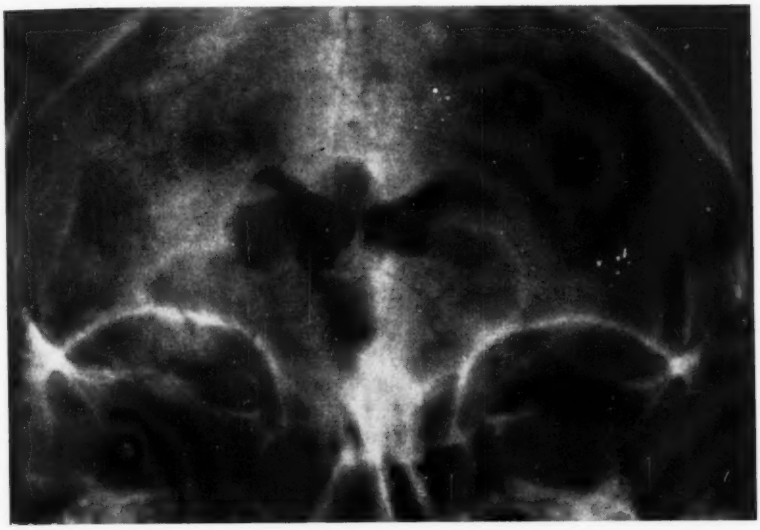

FIG. 1. Case S. G. M. Subfrontal extension of adenoma. Note upward bulge of the floors of both anterior horns. The 3rd ventricle is dilated, deformed and displaced to the right.

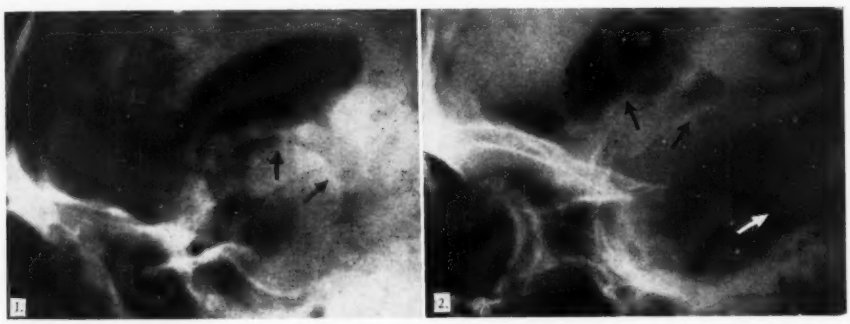

FIG. 2 (*left*). Case C. O. F. Suprasellar extension causing deformation and displacement of 3rd ventricle and of basal cisterns upwards and backwards.

FIG. 3 (*right*). Case E. A. L. Suprasellar extension. Note impression of the floors of the anterior horns. The anterior part of the 3rd ventricle is deformed and displaced upwards and backwards, and the slightly filled interpeduncular cistern is pushed backwards.

dicates that patients with chromophobe adenoma are rarely operated upon only because of their endocrine hypofunctions or general symptoms, the main surgical indication still being the visual deterioration.

In 63 cases (27.6 per cent) the optic discs were normal on both sides or showed only a slight temporal decoloration which could not be diagnosed with certainty as pathologic. Unilateral optic atrophy was found in 48 patients (21 per cent), and bilateral in 117 (51.4 per cent). The postoperative

mortality rate was higher in this latter group (17 per cent) than in the two former (7.9 per cent and 6.2 per cent, respectively).

The early results of operation have been analyzed on the basis of several visual examinations 2–3 weeks after operation. The degree and speed of the visual improvement is related to the preoperative condition of the optic nerves, although a slight decoloration of the papillas is not always a sure sign of atrophy. As described by de Martel, Monbrun and Guillaume,[9] an early decoloration could be the consequence of ischaemia of the optic nerve due to compression of its blood vessels by the adenoma. In these cases the removal of the tumor is followed by a quick recovery because of the normalization of the blood supply, and the vision and visual fields become normal.

Early control examinations were performed on 202 patients with chromophobe adenoma whose visions were found impaired before surgical intervention. Improvement in vision of both eyes was found in 46 (completely normal vision in 11), and of one eye in 36 (normal in 8). Consequently the operation resulted in immediate visual amelioration in 40.6 per cent. Remarkably enough, the number of these cases is exactly the same as the number of cases in which the optic discs showed no pathologic alteration preoperatively or only a slight temporal ischaemia. In 7 cases visual acuity increased on one side and diminished on the other after operation. In 21.2 per cent of the cases surgical intervention caused visual deterioration in both eyes in 25 patients and in one eye in 18. Nine patients became amaurotic in one eye, and 1 in both, due to the immediate effect of the operation; all of them had had very poor vision before surgery. The cause of such blindness is probably the injury of the optic nerves done at the transfrontal manipulation and enucleation of the adenoma, and in some cases suprasellar clot formation. Similar cases were observed by Henderson in the Cushing series only after operations by the transfrontal approach. In 60 cases (29.7 per cent) removal of the adenoma had no effect on the diminished vision in the early postoperative period.

Examination of the patients 2 years or more after operation furnished supportive data for evaluating the late results of surgical intervention. Of the patients who had had chromophobe adenoma, 164 were examined 2 years after removal of the tumor. They were divided into five groups on the basis of their vision: (1) good vision bilaterally (visual acuity 0.8–1.0). There were 35 patients in this group (21.3 per cent). (2) Reduced visual acuity, but still with full ability to work. This group comprises those patients who had moderate loss of vision in both eyes or who had normal vision in one eye but very reduced vision in the other—62 cases (37.8 per cent). (3) Bilaterally impaired vision causing diminished ability to work— 38 patients (23 per cent). (4) Poor vision, finger counting, perception of hand movement, light, etc., and unable to work—23 patients (14.2 per cent). (5) Blindness—6 patients (3.6 per cent).

Consequently, the late results with regard to visual acuity were good or satisfactory in 59.1 per cent. Visual acuity was reduced in 23 per cent. The

patients lost their self-supporting ability in 17.8 per cent. It is rather difficult to compare these data with those of Henderson because he used a different classification based upon the ophthalmological examination as early as the time of discharge from hospital. Later observation, however, gives more reliable and permanent data for judging the final outcome of operation. Nevertheless, the results of our series are in accordance with those of Cushing. Henderson stated that 68 per cent of their patients left the hospital fully able to work as regards vision. Cushing[2] reported that 21 per cent of the transfrontally operated patients regained normal vision and visual field (but only 9 per cent of transsphenoidally operated), which accords with the 21.3 per cent of the first group of our classification.

Acidophil Adenomata. The preoperative examination of 55 patients revealed bilaterally diminished vision in 24 cases (43.6 per cent), unilateral impairment in 19 (34.5 per cent), and normal visual acuity in 11 (21.9 per cent). The optic discs did not show any significant sign of atrophy in 57.4 per cent of the cases; atrophy of one of the papillae was observed in 32 per cent, and bilateral decoloration only in 10.6 per cent. Papilledema of both discs was found in 7 patients, more often than in those with chromophobe adenoma. The postoperative findings of 33 acromegalic patients with impaired vision showed the following distribution: 10 were improved in both eyes (30.3 per cent), 10 in one eye only (30.3 per cent), 11 did not change 33.3 per cent), and further deterioration was observed in but 2 cases. The conclusion is, therefore, that the early results are more favorable in acidophil adenoma cases than in chromophobe. The late results with regard to vision are also better. Two years after removal of the tumor from 29 acromegalic patients, 16 were classified in the first group (55.2 per cent), 11 in the second (37.9 per cent), and 2 (6.9 per cent) had reduced ability to work due only to ophthalmic symptoms. None of the patients became blind or suffered such deterioration of vision that they became unable to support themselves.

The Visual Fields

Chromophobe Adenomata. Of the 232 patients, 147 had bitemporal hemianopia (63 per cent) in the form of complete hemianopia, upper quadrantic defect, temporal constriction or temporal scotoma of the visual fields. That is in accord with Henderson's observations on Cushing's series (65 per cent). Henderson also included in this group, however, those patients with a history of bitemporal constriction of the visual field in whom the visual defect had progressed to blindness in one eye at the time of admission to hospital. Our classification was based entirely upon perimetric findings at the time of surgical intervention.

Removal of the adenomata resulted in complete normalization of both visual fields in 22 patients (14.9 per cent), bilateral improvement in 35 (23.8 per cent), normalization in one eye without change in the other in 13 (8.8 per cent), and unilateral improvement in 8 (5.4 per cent). The constriction of both visual fields progressed after operation in 7 cases and on one side

only in 5. In addition to the 52.9 per cent of amelioration and 8.1 per cent of deterioration, the hemianopia was recorded as unaltered by the operation in 29.5 per cent (43 cases). The postoperative mortality rate of patients with bitemporal hemianopia was 9.5 per cent (14 fatalities).

One eye was blind and the other affected in 36 cases of chromophobe adenoma (15.5 per cent). The operation improved the visual field in 12 cases (33.3 per cent) but did not improve it in 17 (47.2 per cent), and the visual field became worse in 2 cases. There were 5 postoperative fatalities (13.8 per cent).

In 22 cases only one eye was affected by the tumor (9.4 per cent). The effect of the removal of the adenoma in these cases was in general good. Nine patients improved (40.9 per cent); the visual fields of 7 of them became completely normal. The constriction continued in 3 cases; 5 did not change. The postoperative mortality rate was 22.7 per cent (5 cases).

Homonymous hemianopia was present in 7 cases (3 per cent), a rare occurrence in comparison with the 10 per cent of the Cushing series. None of these patients was improved by the operation; in 2 the progression of symptoms could not be arrested, 3 remained unaltered, and 2 died (28.5 per cent mortality).

Normal visual fields occurred in but 9 patients (3.8 per cent). There were no postoperative fatalities in this series. Bilateral blindness was present in 3 patients, of which 1 died. There were 4 instances in which the visual field of one eye was normal and the other eye was blind.

Summarizing these data it can be stated that the best chance for recovery from the visual field defects and the best operative prognosis are offered by the cases with bitemporal hemianopia. Very good results were obtained in those patients who had only upper quadrantic anopia, partial temporal defect or scotoma. The excellent results in patients with normal visual fields are of much less practical importance because of the rare occurrence of such cases. Homonymous hemianopia and other atypical types of defect that indicate deviation of the adenoma from the midline and its lateral extension increase the operative mortality rate and are followed often by disappointing results.

Acidophil Adenomata. Out of 53 acromegalic patients bitemporal hemianopia was present in 22 (41.5 per cent). Extirpation of the adenoma reestablished normal visual fields in both eyes in 8 cases, and improved them in 3 patients bilaterally and in 3 unilaterally (altogether 63.7 per cent). The operation had no effect in 5 cases. There were 3 postoperative fatalities (13.6 per cent).

The visual fields were normal in 8 cases (33.9 per cent); the mortality rate of this series was only 5.5 per cent.

Normal visual field of one eye and temporal defect of the other was observed in 9 cases (16.9 per cent); one improved and the others remained unaltered by the operation.

Homonymous hemianopia was present in 1 case, temporal hemianopia

of one eye with blindness of the other in 2 cases. One of these patients showed a striking improvement on the blind side after removal of the tumor.

ENDOCRINE FUNCTIONS

The endocrine deficiency observed with chromophobe adenomata is due to compression and damage of the pituitary cells by the tumor, and consequently the removal of such an adenoma is rarely followed by improvement in the endocrine functions. The clinical features sometimes become even worse because of the inevitable destruction of the remaining secretory tissue of the gland at the operative manipulation. For this reason, even successful surgical interventions can be followed by complete pituitary insufficiency. However, the removal of an acidophil adenoma, composed of highly active secretory cells, sometimes alleviates symptoms of hyperpituitarism.

Sexual Functions. There were 61 females with chromophobe adenoma between the ages of 15 and 50, and only 9 of them (14.7 per cent) had regular menstruation before operation. The menstruation was irregular in 5 cases and was absent in 47 (77 per cent). Ten patients regained their menses following operation (21.3 per cent of those with preoperative amenorrhea), but only 5 of them regularly and for a longer period. The operation resulted in menopause in 2. Among the 30 female patients suffering from acidophil adenoma there were 8 (26.6 per cent) with regular and 3 with irregular menstruation, and amenorrhea was present in 19 (63.3 per cent). Of the patients with preoperative amenorrhea 16 per cent regained regular menses after removal of the tumor; 1 of them became pregnant several years later and had normal delivery.

There were 119 males with chromophobe adenoma between the ages of 18 and 60 years. Sexual functions were altered in about 46 of them (38.6 per cent) before operation and reduced in 30 (25.2 per cent). Impotence and loss of libido were recorded in 43 cases (36.2 per cent). After operation sexual functions returned in 1 and improved in 2 others; however, in 2 cases they were completely lost. There were 19 males with acidophil adenoma in this age group, and in only 4 instances was impotence recorded (21 per cent); normal function was present in 8 (39.5 per cent) and was reduced in 8 other patients. The operation had no effect upon sexual functions in all but 1 case.

Loss of sexual functions, in Henderson's[6] opinion, is a deprivation syndrome due to compression of the normal anterior-lobe cells by the enlarging tumor, and is not due to the activity of its secretion. Therefore the syndrome is more striking with intrasellar growths, and it occurs later with extrasellar adenomata, which compress the normal pituitary cells only in a later period.

Blood Sugar. It is well known that diabetes is often present in cases of acidophil adenoma (in 25 per cent, as reported by Davidoff and Cushing[4]); blood sugar concentration is usually high and tolerance diminished, as compared with the low blood sugar level of those with chromophobe adenoma. Out of 90 patients with chromophobe adenoma, 80 had a blood sugar concentration below 100 mg. per cent and diabetes was observed in only 1. This

patient had marked diabetes, glycosuria, and a blood sugar level over 200 mg. per cent; however, complete recovery in carbohydrate metabolism occurred after removal of the tumor. On the contrary, diabetes developed in 3 patients, 1, 3 and 8 years, respectively, after enucleation of a chromophobe adenoma.

In 23 cases of acidophil adenoma the blood sugar concentration was examined; it was over 100 mg. per cent in 11, including 5 cases of marked diabetes. One of the diabetics recovered after operation, 1 remained unaltered, while progression of symptoms occurred in 2.

It is almost impossible to analyze the results of the operations with regard to blood sugar changes, because of spontaneous variations and remissions, characteristics of the acromegalic diabetes.

Basal Metabolism. Increased metabolic rate has been observed in many cases of acromegaly (Cushing and Davidoff[3]) but chromophobe adenomata are usually associated with subnormal metabolism. Goiter is a quite common feature in acromegalia; the hyperthyreosis often improves following the removal of an acidophil adenoma. Out of 180 cases of chromophobe adenoma in which basal metabolic rate was determined, 133 (73.9 per cent) revealed a value below 0 (29 of them below −20 per cent). The hypothyroidism in the chromophobe cases is probably due to diminished secretion of thyrotropic hormone by the compressed anterior lobe and, being a deprivation symptom, usually remains unaltered by the operation, sometimes even becoming worse. In such cases surgical intervention can cause myxedema, sometimes years after the operation.

The basal metabolic rates in the acidophil adenoma cases were as follows: in 11 cases below 0 (26.8 per cent), and in 30 above 0 (73.2 per cent), 7 of them being over +20 per cent. Goiter was present in 6 patients.

Blood Pressure. Among the patients with chromophobe adenoma (224 examined), 66 (29.4 per cent) had a systolic pressure between 80 and 120 mm., 132 (58.8 per cent) between 120 and 160, and 26 (11.8 per cent) above 160 mm. On the other hand, of the 52 with acidophil adenoma only 10 (19.2 per cent) had a pressure below 120 mm., while in 20 (57.7 per cent) it was between 120 and 160, and in 12 (23.1 per cent) above 160 mm.

Removal of the tumor does not influence the high blood pressure. It remains unaltered in some cases during the following years; frequently, however, it shows progression, especially in cases of acidophil adenoma, sometimes leading to death from cerebral hemorrhages.

LATE RESULTS OF THE OPERATIONS

Chromophobe Adenomata. Accurate follow-up of almost all patients who lived in the countries of Scandinavia has been possible, and examinations, or reports written by their doctors or themselves, have been obtained yearly. However, records of the late results of those who came from other countries have sometimes been incomplete, especially during the years of war.

Of the patients with chromophobe adenoma who were operated upon in

the past 18 years and survived the operation, 130 are still living, 9 of them after further operation for recurrence of the neoplasm. The death of 40 patients was reported: 8 died from recurrence of the tumor, 5 from endocrine insufficiency, 18 from incidental causes, and 9 from unknown conditions. It was possible to follow 176 patients over 2 years after removal of the adenoma. The ability to work and the question of self-support was determined individually, the age and profession of each patient being taken into consideration. There were 96 fully able to work (54.5 per cent) after the 2-year period. Representatives of various mentally and physically exerting professions were found in this series (engineer, navy officer, horseman, sportsman, woodcutter, etc.), demonstrating that a successful surgical intervention can be followed by complete mental and physical restitution. The ability to work was reduced in 48 cases (27.2 per cent) for various reasons, in 33 being due to loss of vision. Reduced vision was reported also by some other patients in

TABLE 3

Survival Period of Patients with Chromophobe Adenoma

Elapsed Time after Operation	Maximum Number of Patients	Untraced	Alive	Per Cent Alive	Died
2 years	195	13	147	80.8	35
4 years	158	11	111	75.5	36
6 years	126	14	89	79.4	23
10 years	74	10	49	76.5	15
14 years	22	1	12	57.1	9
18 years	2	0	1	50.0	1

whom the reduced ability to work was due mainly, not to reduction in vision, but to other reasons—in 6 cases, to impaired mental condition, weakness, or indifference; in 4, to hypertension; in 2, to epileptic seizures; in 2, to endocrine disturbances (myxedema, diabetes), and in 1, to persistent strong headaches. Inability to be self-supporting was reported by 32 patients (18.3 per cent)—in 24 cases due to loss of vision or blindness, in 6, to mental symptoms, headaches, and apathy, in 1, to cerebral hemorrhage, and in 1, to frequent seizures.

It has been supposed that the long postoperative survival periods in our series (Table 3) are due to the transfrontal enucleation of the adenomata. Although this type of operation has a higher mortality rate, it represents a much more radical removal of the neoplasm and consequently there are fewer late fatalities from recurrence than after the transsphenoidal operation. Furthermore, another important factor in the long survival periods is the postoperative x-ray treatment which was given routinely with but very few exceptions. A definite improvement in the late results of operation when followed by x-ray treatment was described by Henderson.

Acidophil Adenomata. Of the patients operated upon for acromegalia, 19

have been reported to be still alive and 14 others died. The cause of death was splanchnomegaly and dilatation of the heart in 4 cases; hypertension, arteriosclerosis, cerebral hemorrhage in 3, diabetes in 2; recurrent tumor in 2; and progressing pituitary insufficiency and adynamia in 1 (autopsy revealed an old clot at the site of the pituitary body and hemorrhages in the hypothalamus). In 2 cases the cause of death was not reported. Death due mainly to diabetes was not observed so often as in the Cushing series, in which 6 such fatalities and 7 due to cardiovascular changes were reported.

Among the 30 patients with acidophil adenoma who were followed up, 17 were found fully able to work (56.6 per cent), and 2 were alive without any symptoms but did not report exactly about their ability. There was a reduced ability in 7 cases (23.3 per cent), mainly because of fatigue and headaches (3), hypertension (2), and visual failure (2). Four patients were unable to work (13.3 per cent), 3 of them because of progressing acromegaly, fatigue and headaches and 1 on account of epileptic fits.

TABLE 4

Survival Period of Patients with Acidophil Adenoma

Elapsed Time after Operation	Maximum Number of Patients	Untraced	Alive	Per Cent Alive	Died
2 years	44	3	31	75.6	10
4 years	37	4	21	63.6	12
6 years	33	3	17	56.6	13
10 years	18	5	7	53.8	6
14 years	2	—	0	0	2

In contrast with the chromophobe adenoma cases, visual failure is rarely the cause of postoperative inability in acromegalics. Usually there is a marked variation in intensity of the acromegaly after operation; active and inactive periods alternate, with intervals of shorter or longer length, and sometimes with spontaneous remissions. The prolonged follow-up revealed that acromegaly progressed without remission in all but 5 cases. In these 5 patients the symptoms did not advance; there was even a slight improvement in 3 of them due to remission of the hypertrophic soft tissues, especially on the face. Similar observations were made in fugitive acromegaly of patients with chromophobe adenoma, which frequently disappeared after removing the tumor.

The periods of survival of acromegalic patients are recorded in Table 4. These data are much less favorable than those of the chromophobe adenomata, although the average age of acromegalics is lower and there are fewer old individuals among them. A considerable percentage of the patients died after an interval of 4 years or more after operation.

RECURRENCES

Thirteen patients (12 chromophobe and 1 acidophil) were operated upon again for recurrence of symptoms; that represents 5.2 per cent of those

operated upon for chromophobe adenoma and 1.8 per cent of the acromegalics. The total number of surgical interventions was 16, as one patient was operated upon three times for recurrence, and another twice. To this number of recurrences 5 other chromophobe and 4 acidophil adenomata can be added; in these cases recurrences were proved by evident clinical symptoms or by autopsy. It is hardly possible to give exact percentile rates with regard to recurrences of pituitary adenomata, because they can occur many years after the primary operation. However, reliable data can be obtained from that part of the series that has been followed up carefully and in which at least 5 years have elapsed since removal of the tumor. By such standards a rate of recurrence of 10–15 per cent has been estimated for the chromophobe adenomata and 20 per cent for the acidophil. The effect of prophylactic x-ray treatment could not be analyzed on account of the lack of untreated controls, postoperative radiation having been given in nearly all cases.

Out of Cushing's 247 patients with chromophobe adenoma, 44 were operated upon again for recurrence and 70 others died from recurrences (together 46 per cent). In the acidophil adenoma group the rate of recurrence was 37 per cent. The far fewer recurrences in our series indicate the advantage of a transfrontal operation from the standpoint of recurrence.

The symptoms returned usually within a few years after the primary operation, but some exceptions were recorded. Careful analysis of 20 cases revealed that the first symptoms of recurrence appeared in 2 patients within 1 year, in 6 within 2 years, in 1 within 3 years, in 5 within 4 years, in 3 within 5 years, in 1 within 6 years, in 1 within 7 years, and in 1 within 8 years after the first operation. Consequently, in a vast majority of the cases the recurrence is to be expected within 5 years after operation. The first sign is mainly visual failure, in many cases a rather rapid loss of vision; it was observed in 90 per cent of recurrences. But other symptoms can cause suspicion, too, varying according to the pathway of extension. Hypothalamic symptoms (drowsiness, polydipsia, polyuria) were observed in 5 cases, headaches in 8, epileptic fits in 1, and mental disturbance in 1. Sexual functions that have remained or improved after the primary operation can be lost again; amenorrhea or impotence was recorded in 4 cases. In 3 patients the chief sign of recurrence was the progression of their cavernous sinus syndrome. In 1 of these, ptosis of the left eyelid had been noticed before the first operation, and it improved considerably after removing the adenoma. Eight years later examination revealed total ophthalmoplegia on the same side and palsy of the trochlear nerve. The recurrent tumor was enucleated and a marked improvement has been recorded.

There was no histological evidence of malignancy, change in chromatin structure, polymorphism or increased mitotic activity in the recurrent adenomata; the microscopic picture was that of the usual chromophobe adenoma. The structure of the recurrent neoplasm was usually completely identical with that of the first tumor. This is rather striking in those 2 patients whose adenomata revealed a marked tendency to grow and to return. One of them had recurrences and repeated radical enucleations 4, 6, and 8

years after the primary operation. Walnut- and hazelnut-sized tumors were removed each time. Microscopically there was no evidence of malignancy, but the abundant blood supply of the adenoma was striking on all occasions. The cells were in alveolar arrangement around the coherent network of capillaries. They had a large protoplasm of highly cylindrical shape, and secreted colloid in abundance, which was deposited in the stroma in form of hyaline masses. The other patient had recurrences 2 and 5 years after the first operation. Each time the adenoma showed a somewhat papillary structure, but no polymorphism was found. On the basis of this evidence there is no histological explanation regarding the potency of growth or tendency to return of these tumors.

The removal of recurrent adenomata usually is not more complicated than the primary operation, and the transfrontal approach can be repeated several times without increased risk. The mortality rate of secondary procedures was even less, only 6.2 per cent. The expectation of patients who have had reoperations is not bad, although the late results are less favorable than those of patients without recurrence, especially regarding vision. Late examinations revealed that none of these patients regained full visual acuity or complete visual fields; there was moderate visual failure in 5 and severe loss in the other 5. Three patients regained full ability to work following the repeated interventions, but the ability remained reduced in 5, was minimal in 1, while 1 was not capable of self-support. The time of survival was not less than of those without recurrences; one patient died 2 years and another 3 years after the secondary operation, but the others are alive after the repeated intervention—4 after 2 years, 3 after 6 years, 1 after 7 years, 1 after 9 years, and 1 after 10 years.

SUMMARY

During the period 1929 to 1947, 292 patients with verified pituitary adenoma were treated and 300 operations performed. The series included 232 chromophobe, 55 acidophil, 2 basophil, and 3 malignant adenomata.

The postoperative mortality rate was 11.3 per cent for the chromophobe and 9.5 per cent for the acidophil adenomata. There was a striking difference, however, between the group of intrasellar tumors and the group with extrasellar extension, the first having a mortality rate of 6.4 per cent, and the second, 35.0 per cent.

Cases of adenoma with extension in the cavernous sinus, cave of Meckel, temporal lobe, hypothalamus, or third ventricle were included. Those in which there was extension in the cavernous sinus did not reveal a higher postoperative mortality rate than cases of the usual adenoma. There were more fatalities among patients with hypothalamic signs, and the late results of operation were not favorable.

The ophthalmic and endocrine symptoms and the effect of the removal of the pituitary adenomata thereon are discussed.

Two years after operation or later, 54.5 per cent of the surviving patients

with chromophobe adenoma were fully able to work; the ability was reduced in 27.2 per cent of the cases, and lost in 18.3 per cent. Among the acromegalic patients the late postoperative ability to work was complete in 56.6 per cent, reduced in 23.3 per cent, and lost in 13.3 per cent. The radical transfrontal removal of the adenomata and the postoperative x-ray treatment in this series were followed by long survival periods and relatively few recurrences.

There were 16 operations for recurrence of the adenoma. The mortality rate after secondary procedures was less than that after primary operations, and the time of survival was usually long. The late results, however, were less favorable in regard to ability to work. Recurrence rates of 10 to 15 per cent for the chromophobe and about 20 per cent for the acidophil cases are estimated.

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CLINICAL USES OF AN ARTIFICIAL CEREBROSPINAL FLUID

R. C. LEWIS, M.D., AND K. A. C. ELLIOTT, PH.D.
Department of Neurology and Neurosurgery, McGill University, and the Montreal Neurological Institute, Montreal, Canada

(Received for publication October 11, 1949)

VARIOUS solutions have been used to irrigate the brain and to replace lost cerebrospinal fluid. It has been amply demonstrated by Flexner and Amoss,[3] Weed and Wegeforth,[6] and Kasahara[4] that the more nearly the replacing fluid resembles CSF, the less the reaction measures clinically or by cell and protein studies. The solution should have the proper inorganic salt content, pH, and osmotic pressure. It must be free from pyrogens and not be altered by sterilization. Finally, it should be easy to handle and not be liable to contamination. Autologous CSF has two disadvantages. One is that sterility is difficult to maintain for long periods. Secondly, on exposure to air the pH changes in the alkaline direction. Sachs[5] observed reactions with autologous fluid and believed that they were due to the contained protein, though it seems possible that developing alkalinity was responsible, since precautions to prevent loss of CO_2 and consequent developing alkalinity were not mentioned. He used a bicarbonate buffered artificial CSF prepared by Hartmann and stated that the reaction was less marked.

One of us has devised a solution similar to Hartmann's but which is much easier to prepare. We have found it especially useful clinically in the treatment of hydrocephalus and of subdural hematomas.

The solution in question was described by Elliott and Jasper[2] (their Solution B), and its method of preparation is outlined in the appendix to this paper. It contains inorganic salts, including bicarbonate, and glucose, in concentrations similar to those in CSF and has the pH and osmotic pressure of normal body fluids.

In hydrocephalus, complete replacement of ventricular fluid with oxygen is often necessary for exact localization of the defect or obstruction in the absorbing pathway. Davidoff and Dyke[1] pointed out the reaction of the central nervous system to pneumography, which was in proportion to the amount of injected air or oxygen. In 4 cases, we have replaced the ventricular oxygen on 5 occasions. A ventricular needle is placed in each ventricle, and the fluid at body temperature is introduced through the lower needle from the reservoir of an infant transfusion set (Fig. 1). A set of typical roentgenograms illustrating the completeness of the ventricular fluid replacement is shown in Fig. 2. Following these procedures, the "artificial spinal fluid" having been used as replacement fluid, reactions were minimal. The child is apt to vomit the first day. Temperature elevations from 100.4°

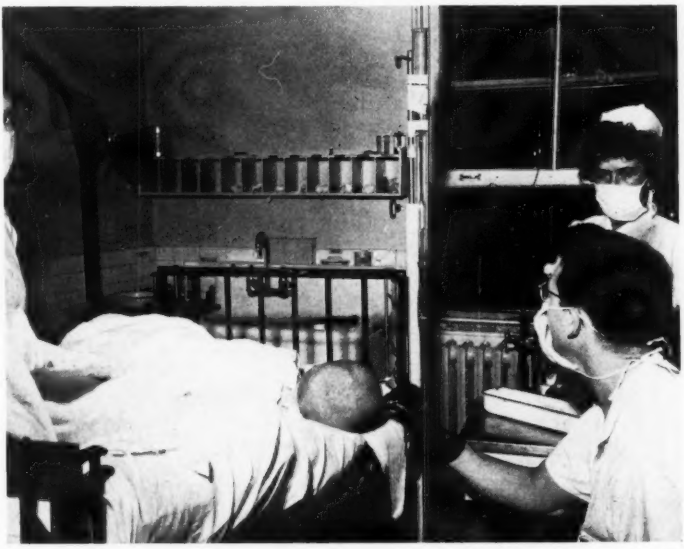

FIG. 1. Ventricular fluid replacement by the 2-needle method. The procedure is carried out in the surgical dressing room with the aid of two nurses.

to 102.2° have occurred but have not lasted more than 1 day. Within the first few days the intracranial pressure may become elevated, so that a ventricular tap is required. In these cases, the protein has been not over 16 mg. per cent and cell counts have varied from 0 to 600. In 1 case 850 cc. of

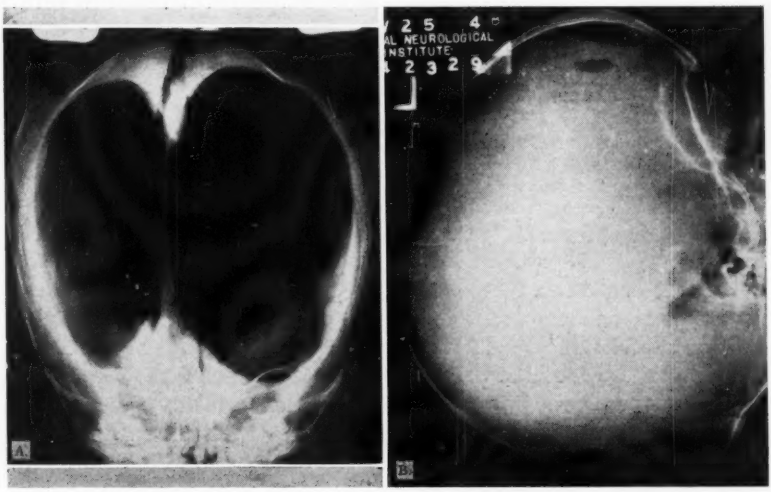

FIG. 2. Representative roentgenograms from a case of hydrocephalus demonstrating the completeness of ventricular fluid replacement (B) following ventriculography (A).

buffered solution were used without untoward effects. In Table 1 data concerning these cases are presented.

After removal of a large subdural hematoma, or a large intracranial tumor, the brain may fail to expand, leaving a dead space (Fig. 3). We feel it is desirable to obliterate this cavity by gently introducing some innocuous fluid into the spinal subarachnoid space. In the past we have injected air at the close of the operation while expansion of the brain could be followed by

TABLE 1

Reactions to Artificial Cerebrospinal Fluid Replacement After Ventriculography

CASE	AGE	WEIGHT	AMOUNT OF REPLACEMENT FLUID	CLINICAL REACTION	NUMBER OF VENTRICULAR TAPS	C.S.F. REACTION	RECTAL TEMPERATURES (ARROW SHOWS TIME OF THE PROCEDURE)
R.A.	5 WKS.	7¾ LBS.	150 c.c.	VOMITED 1 OZ. FROM EACH OF FIVE FEEDINGS.	ONE (4TH DAY)	—	
S.M.	8 MOS.	22 LBS.	850 c.c.	VOMITED ONE FEEDING.	ONE (1ST DAY)	PROTEINS: LESS THAN 11 CELLS: NONE	
E.C.	8 MOS.	21 LBS.	360 c.c.	NO VOMITING	ONE (1ST DAY)	PROTEINS: LESS THAN 11 CELLS: NONE	
C.S.	21 MOS.	21 LBS.	450 c.c.	VOMITED TWO FEEDINGS. NECK STIFF ONE DAY.	TWO (1ST DAY) (3RD DAY)	PROTEINS: 16 CELLS:600 POLYS. CULTURE: NEG. CELLS:100 POLYS. 100 LYMPHS.	
C.S.	22 MOS.	21 LBS.	750 c.c.	VOMITED FIVE FEEDINGS	NONE		

direct vision. The air has, however, provoked some signs of meningeal irritation. For that reason, a modified, bicarbonate-free Ringer solution (Solution A of Elliott and Jasper[2]) was used at first; later the artificial CSF was used.

During the past year, there were 5 cases of subdural hematoma in which the brain did not re-expand spontaneously. Artificial CSF was used in 2 cases, the modified Ringer solution in 2, and air in 1. Temperature responses following use of the artificial CSF were only 101° whereas the other methods resulted in temperatures from 102° to 102.6°. One patient treated with the artificial CSF had 8 cells in the CSF the following day; the other had 340 WBC associated with 32,000 RBC. The only other cell count obtained was in the case in which oxygen was used: there were 480 polymorphs, 90 lymphocytes, and 22,500 RBC the day following operation.

In a recent case of low-pressure headache following a myelogram, the headache was relieved instantly by intrathecal injection of 50 cc. of the artificial CSF. The headache returned in 24 hours but was still noticeably improved. CSF cell counts before the procedure and then 24 hours after the procedure were respectively 38 and 10 lymphocytes.

In summary the bicarbonate buffered "artificial cerebrospinal fluid" developed in this Institute, which is easy to prepare, has caused only minimal

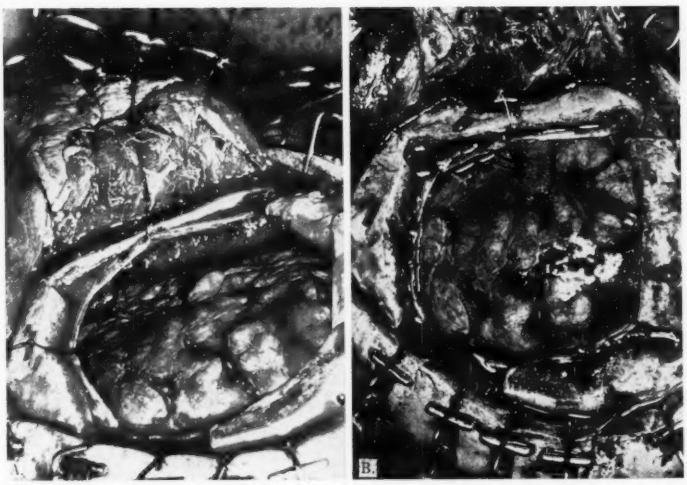

FIG. 3. Photographs taken at operation after removal of a large subdural hematoma. The distance from brain to dura is shown before (A) and after (B) intrathecal injection of "artificial cerebrospinal fluid."

reactions either in replacing ventricular oxygen, or in re-expanding the brain to fill the cranial space.

We would like to express our appreciation of the encouragement of Dr. W. V. Cone under whose direction this work was done and from whose service the clinical records were obtained.

(*See next page for Appendix*)

APPENDIX

Preparation of "Artificial Spinal Fluid"

Alkaline-Salt Solution

Stock Solution

$NaCl$	236 g.
Na_2CO_3	92 g.
KCl	12 g.
$Na_2HPO_4 \cdot 12H_2O$	8 g.
Phenol red 1 mg/ml	4 ml.

(Dissolve 100 mg. phenol red in 5 ml. 0.1 N NaOH and dilute to 100 ml. with 25 per cent NaCl solution)

Dissolved, filtered through sintered glass, and made to 1 l. with distilled water.

Use Solution

25 ml. of the stock solution are diluted with distilled water to 1 l.

Acid-Salt Solution

$CaCl_2$	24 g.
$MgCl_2 \cdot 6H_2O$	12 g.
Concentrated HCl	300 ml.

This solution is self-sterile. It is kept in a bottle with a helmet type cap and away from surgical instruments.

Glucose Solution

Dextrose (anhydrous)	8 g.
Water	500 ml.

The alkaline-salt and glucose solutions are autoclaved in convenient quantities. Acid-salt solution is added to the alkaline-salt "use" solution—a little more than 2 ml. of the former per l. of the latter—with thorough mixing, until the pink color is discharged. This addition is best made by means of a sterile syringe with a thin glass tube, instead of a needle, attached by rubber tubing. In view of the strong buffering capacity of the bicarbonate, exact measurement of the acid solution is not critical. The sterile glucose solution, 50 ml. per l., is added and the whole well agitated. The mixture should be prepared shortly before use; it should not be warmed above about 55°C. and should not be exposed in an open container for long periods.

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THE LONGEVITY OF PATIENTS WITH GLIOBLASTOMA MULTIFORME

MARTIN G. NETSKY, M.D., BURTON AUGUST, M.D., AND WILLIAM FOWLER, M.D.*

Laboratory Division, Montefiore Hospital, New York, N. Y.

(Received for publication October 24, 1949)

THE ALMOST uniformly short life span of patients with glioblastoma multiforme is well known. Many neurosurgeons have based their handling of such patients on this fact. It is therefore of interest when patients with long survival are found. In a review of gliomas at Montefiore Hospital, a few cases were encountered in which the duration was so long that they were deemed worthy of being placed on record. Stimulated by this finding, we have attempted an analysis of factors in prognosis.

MATERIAL

The total number of gliomas reviewed was 165. Of these, 83 (50.4 per cent) were glioblastoma multiforme. For purposes of this presentation, 70 cases were considered. The remainder were omitted mainly because the follow-up was inadequate. Within the group of 70, there were 59 patients who came to necropsy, and 11 in whom the diagnosis was proved by biopsy alone. There were 57 patients upon whom operation was performed, many of these later coming to necropsy. Of these 57 patients, 35 received radiotherapy in addition, and 2 received courses of nitrogen mustard. Of the patients who were not operated upon, only 1 was irradiated.

There was a marked difference in incidence between the sexes; 45 of the patients were males, and 25 females, a ratio of 1.8:1. The age of onset reached its peak in the 6th decade. The youngest patient was 19, the oldest 70. In terms of location of the neoplasm, all patients had some cerebral involvement, but in 1 case the tumor extended in the mid-brain, and in 1 other case the tumor was predominantly metencephalic.

The survival times measured from the first identifiable symptom or sign are shown in Fig. 1. The average duration from the first symptom to exitus was 17 months. However, this average was distorted by the unusual cases to be discussed. The median survival was 8.3 months, a figure that is more indicative of the usual case. Expressed another way, almost 80 per cent of the patients died within 20 months of their first symptom, and 90 per cent within 30 months.

The longest survival was 14 years. There were 5 patients who survived 6 years or longer. In 2 of the latter cases, biopsies were performed early in the course. Since they are representative of the group, they will be described in detail.

* Present address: 901 Vancouver Block, Vancouver, B. C.

CASE REPORTS

Case 1. S.S., Autopsy #9588. *Onset with focal motor seizures. Removal of malignant glioma 5 years later. Normal life for many years. Death with autopsy 15 years after onset. Glioblastoma multiforme.*

In 1928, at the age of 37, this right-handed white male had a convulsive seizure lasting a few minutes. Thereafter at intervals of about 2 to 3 months, he had attacks beginning with numbness and twitching of the right hand, then a tonic convulsion of the right side and subsequent loss of consciousness. In 1931, following an attack, he noticed loss of sensation on the right side of the body and a right hemiparesis. In 1933 a partial motor aphasia developed.

Operation. Dr. Paul C. Bucy operated on him in October, 1933. A firm area meas-

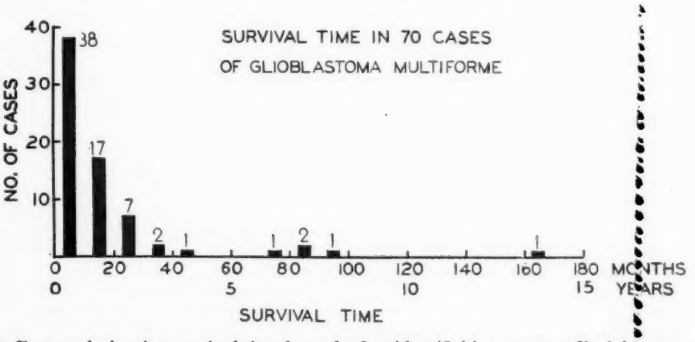

FIG. 1. Bar graph showing survival time from the first identifiable symptom. Each bar represents the number of patients surviving for a 10-month period. Note that there are 5 patients with survival of more than 5 years.

uring 2 cm. in diameter was found on the left side in the gyrus just anterior to the precentral gyrus. The remainder of the brain in the operative field appeared normal, except for very slight flattening of the convolutions. On incision the tumor was tough, and reddish-gray in appearance. An attempt at complete removal was not made because of the location. A small piece was taken for study, and a temporal decompression performed.

Microscopic Examination. The tissue was diagnosed by Dr. Percival Bailey as a malignant glioma, probably a glioblastoma multiforme. The slide was studied by us and this diagnosis confirmed. It should be emphasized that many mitotic figures were present. Fig. 2 illustrates the character of this neoplasm.*

Course. Following the operation, the patient was given a course of roentgen therapy for a total of 6,068 r. and was placed on phenobarbital. For the next 4 years he was free of convulsive attacks. His control over the right side of the body improved, and he was able to write and use tools. He gained in weight and general health. In September, 1937, he was struck by an automobile, and was unconscious for 24 hours. Sometime thereafter his seizures returned. He gradually lost power on the right side, and aphasia became worse.

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* We are indebted to Dr. Theodore Rasmussen for permission to examine the slides and reproduce the picture.

In October, 1939, he was seen by Dr. Leo Davidoff. At this time the findings were those of right-sided weakness, slightly hyperactive deep reflexes on the right, and a concave left temporal decompression. An encephalogram was interpreted as showing left-sided cerebral atrophy without evidence of regrowth. Nothing further was done.

He entered Montefiore Hospital in August, 1940 at the age of 49. In addition to the above findings there were now astereognosis in the right hand, a partial motor aphasia, and some blurring of the right disc margin. The CSF contained 3 cells, with 52 mg. per cent protein. The Wassermann test was negative. During his stay in the hospital, both focal motor and generalized seizures were noted. Irradiation was not deemed advisable, and he was discharged to be followed in the out-patient clinic.

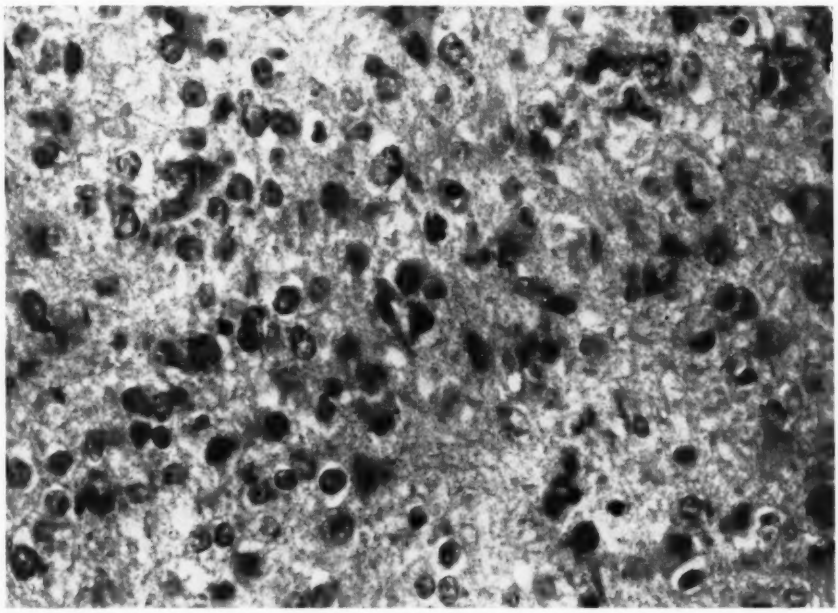

FIG. 2. Case 1. Photomicrograph of specimen from original biopsy to demonstrate glial cells and mitotic figures. Hematoxylin and eosin stain, ×425.

He had increasing difficulties in expressing himself. Because of this, he insisted on being re-admitted in July, 1941. At this time it was felt that the paretic side was more spastic, and that the motor aphasia now had a sensory component. CSF examination, including cells, protein, and pressure, were within normal limits. He was given a course of roentgen-therapy amounting to 3,000 r. with slight improvement. Thereafter he continued to have right-sided seizures until July, 1943, when he rapidly became worse and died. A lumbar puncture 5 days before death revealed a pressure of 560 mm. of water.

Autopsy. A broncho-pneumonia was found to be the immediate cause of death. The brain weighed 1,420 gm. The left hemisphere was slightly larger than the right.

The frontal convolutions were flattened. There was a hemorrhagic tumor nodule, measuring 3 × 2 cm., in the left 3rd frontal convolution. Other small hemorrhagic nodules were seen in the left precentral gyrus and the superior temporal gyrus. The cerebellum and brain stem were shifted slightly to the right. Sections of the frontal lobes disclosed a large hemorrhagic and necrotic neoplasm (Fig. 3). It measured 5

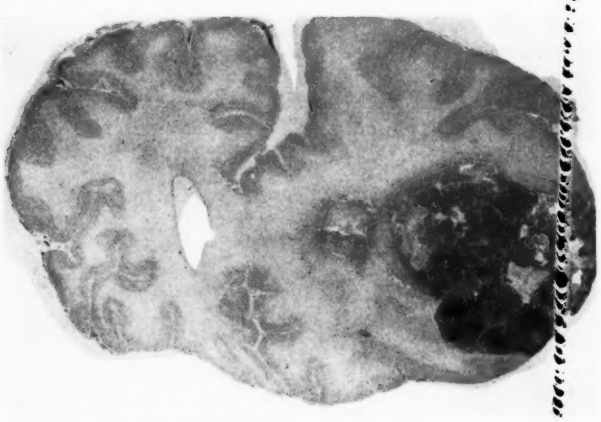

FIG. 3. Case 1. Coronal section through the frontal lobes. The neoplasm is hemorrhagic, the left hemisphere swollen, and the midline shifted. Nissl stain.

by 3 cm. and involved both white and gray matter. The left lateral ventricle was constricted and pushed to the right. The tumor disappeared from view when the basal ganglia were reached, but there was swelling of the white matter throughout the left hemisphere. The left putamen contained a hemorrhagic cyst. The cortical architecture in the left premotor convolutions was distorted.

Microscopic Examination. The tumor was a glioblastoma multiforme. There were some spongioblastic portions, but there were, in addition, extensive necrosis, pseudopalisading around focal necrotic zones, thrombosis of vessels and endothelial proliferation, perivascular lymphocytes, numerous mitotic figures, and pleomorphism (Fig. 4).

Comment. This patient had a histologically malignant neoplasm at the time of his operation. This was equally true of the tumor seen at autopsy. The length of survival is, to the best of our knowledge, the longest on record in a proved case of glioblastoma multiforme.

Case 2. B.K. Autopsy #10,210. *Onset with vomiting, dizziness, and alteration of voice. Operation 1 year later with removal of cerebellar astrocytoma. Death with autopsy 7 years after onset. Glioblastoma multiforme.*

In 1938, at the age of 24, the patient, a right-handed white female, experienced "morning sickness." After awaking, she would vomit a small amount of green fluid and then felt better. Several weeks later, she noticed dizziness occurring 2 or 3 times a week. This was associated with a feeling of unsteadiness in the legs, and a tendency to fall to the right. These bouts lasted about 10 minutes. At this time, her voice changed, and there was occasional gasping. The morning vomiting, dizziness, and

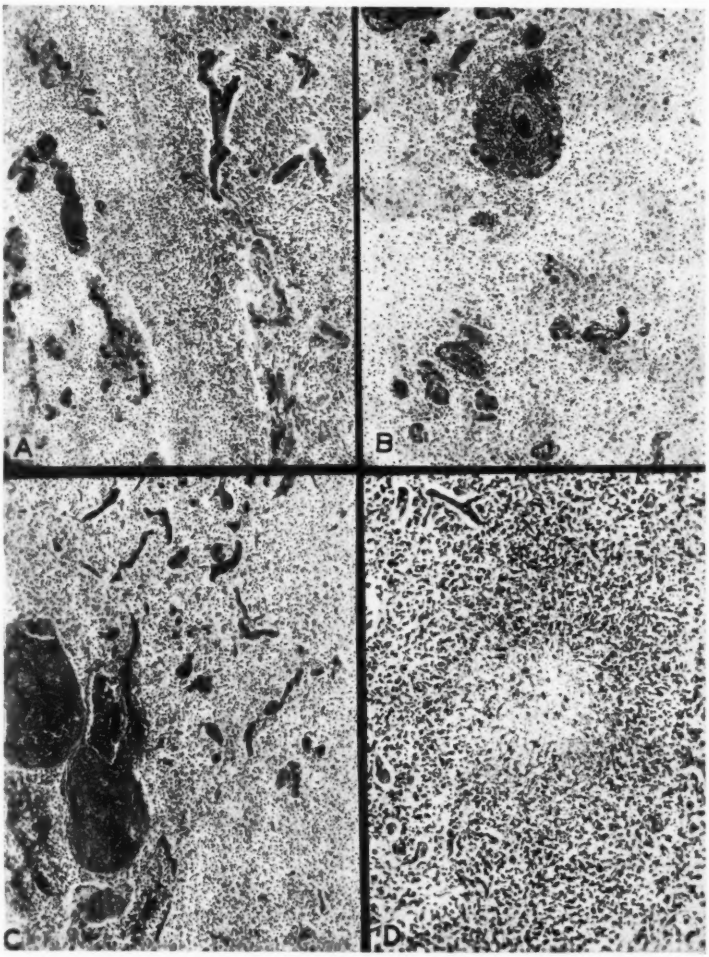

FIG. 4. Case 1. (A) Some spongioblasts are on the right. There is partial necrosis of tissue on left. ×93. (B) Perivascular lymphocytes surround some of the proliferated vessels. ×93. (C) There is thrombosis of blood vessels and vascular proliferation. ×93. (D) A zone of focal necrosis with pseudo-palisading. ×120. All sections are stained with hematoxylin and eosin.

voice difficulties became steadily worse. In September, 1938 her right foot began to drag. In November she had difficulty using her right hand and arm. Swallowing difficulties supervened and regurgitation of food occurred. In December she entered a hospital where the CSF was at a pressure of 140 mm., and the Wassermann, colloidal gold and globulin tests were negative. A right recurrent laryngeal paralysis was found. The clinical diagnosis was a posterior fossa lesion. She was found to be pregnant and was aborted in January, 1939.

Operation. In February, 1939 a craniotomy revealed a cerebellar and brain stem neoplasm, which was biopsied.

Microscopic Examination. A diagnosis of astrocytoma was made. The diagnosis was confirmed at this hospital.*

Course. Her speaking and swallowing improved, but the dizziness and vomiting continued. She received a course of x-ray therapy and was admitted to Montefiore Hospital in July, 1939. Here she was given further x-ray therapy and discharged to be readmitted in June, 1942. At that time she stated that she had been able to get

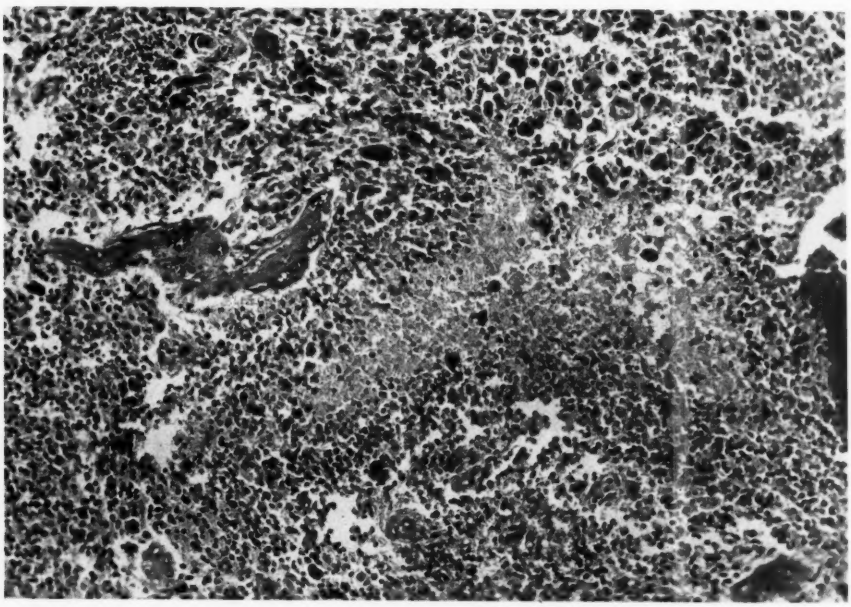

FIG. 5. Case 2. Focal necrosis, pseudo-palisading, endothelial proliferation, giant cells and pleomorphism are shown. Hematoxylin and eosin stain, ×88.

about fairly well, except that 2 months prior to admission, swaying and weakness increased. Another series of radiation treatments was given. Her final admission was in July, 1945, when she entered complaining of severe occipital headaches and some diminution of vision. Examination at this time showed the presence of cerebellar signs on the right, and palsies of the right 3rd, 6th, and 7th cranial nerves. Her course was then progressively downhill. She became cyanotic, comatose, and died in July, 1945.

Autopsy. Examination was limited to the nervous system. Externally, the neoplasm was seen in the right cerebellar hemisphere and the right side of the pons and medulla. On sectioning the brain, the corpus callosum was enlarged and contained numerous cysts. The right side of the pons was replaced by a white, firm tumor with

* We wish to thank Dr. Joseph Felsen for permission to examine these slides.

no sharp boundary between it and normal tissue. There was extension of the tumor to the left side of the pons. The neoplasm in the right cerebellar hemisphere was hemorrhagic. A separate neoplasm of similar appearance was present in the left cerebellar hemisphere.

Microscopic Examination. The cysts in the corpus callosum were astrocytic. The cerebellar and pontine neoplasms were typical glioblastoma multiforme (Fig. 5).

Comment. In this case it was less certain that a malignant neoplasm was present from the onset. It was possible that the biopsy was taken from the edge of a deeper-lying, more malignant glioma. Another interpretation was that a more benign neoplasm became malignant spontaneously, or following operation and irradiation. It is not possible to determine which alternative explanation is correct. Nevertheless what is deemed a malignant neoplasm histologically may sometimes be compatible with a relatively long survival. Case 1 represents more certain evidence of this.

DISCUSSION

The following factors have been considered as possible aids in determining survival in cases of glioblastoma multiforme.

1. *Age of Onset.* The longest survivals (6 years or more) all occurred in patients whose age at onset was from 24 to 42 years. There were many others, however, in this age range who had only the usual brief survival period. None of the older nor the younger patients had a long survival. It should be noted that the peak of the age-incidence curve of glioblastoma is at 50–60 years.

2. *Sex.* There is a definitely higher incidence of glioblastomas in males. This had been found by many others, see for example, Busch and Christensen.[2] Because of the higher incidence, the question was asked whether survival was shorter in males. In this series there was no significant difference in the average survival time of the two sexes.

3. *Initial Symptoms.* The average survival of all patients was 17 months. In 13 patients in whom a motor seizure of any type was the first symptom, the average duration was 39 months. If the seizure was focal motor in character (9 cases), the average survival was lengthened to 51 months. Unfortunately, the focal seizure was not uniformly indicative of greater length of survival. Table 1 demonstrates that 3 of the 9 patients in this group had a survival around the general average. There was nothing in the succeeding course that could be correlated with length of survival. Bailey and Cushing[1] reported 5 of 77 cases with survival of over 3 years from the onset of symptoms—3, 4, 6, 6, and 12 years. They commented that "the longest survival periods all prove to be ones with a premonitory history of Jacksonian seizures for many years before pressure symptoms supervene." While this was not invariably true in our long-surviving cases, longer life with this initial symptom is a striking fact. The Montreal group reported 3 cases with long survival and an early history of focal epilepsy.[3]

It is not clear why this should be true. Since operation appears to have

little effect on longevity (see below), it cannot be explained by the fact that a seizure brings the patient sooner to the neurosurgeon.

4. *Operation.* In 57 of 70 cases, operation was performed. A variety of types of internal and external decompressions were done. Symptoms were present an average of 7.1 months before operation, and the average postoperative survival was 10.7 months. Therefore, the average survival from the first symptom was 17.8 months, although the median was 13.9 months. The average duration of non-operated cases was 12 months. The average duration of all cases in the series was 17 months. It therefore appears that operation alone does not significantly increase longevity. Decompression may afford the patient more comfort but life is not prolonged.

TABLE 1

Survival in Cases with Focal Motor Seizure as the Initial Symptom

Patient	Survival from First Symptom in Months	Location of Tumor	Number of Operations	Postoperative Survival in Months	Irradiation
S.S.	168	Left F-P	2	120, 12	Yes
H.K.	81+	Right T-P	2	24+, 12+	Yes
F.K.	74	Right T-P	2	49, 7	Yes
A.S.	48	Left F-T-P	3	18, 18, 7	Yes
G.S.	24	Left T-P	1	24	Yes
L.S.	20	Right F-P	1	17	Yes
M.M.	18	Right F-T-P	2	13, 11	Yes
C.C.	14	Right F-P	1	6	Yes
N.K.	13	Left F-T-P	1	4	No

Note: The location in H.K. and F.K. was determined by biopsy only. H.K. is alive at the present time. All other patients in this group came to necropsy.

5. *Irradiation.* Among the 57 patients operated upon, 35 received radiation postoperatively. The details of therapy were so varied that it would be futile to subdivide them. The average survival from the first symptom, in the irradiated group, was 27 months, median 13.1. The average survival in the non-irradiated group was 8.8 months, median 6.2. Within the latter group, however, were so many cases in which death occurred in the immediate and later postoperative period that the average gives a false impression. Thus, eliminating those patients who died within 1 week after operation, the average survival of non-irradiated patients is raised to 12 months. It appears that patients who live longer have more opportunity to be irradiated rather than the reverse. The effect of the usual course of irradiation on longevity is slight.

6. *Location.* An attempt to correlate longevity with location of the tumor within the cerebrum yielded no significant results. This in part may have been due to the predominance of autopsy cases in this series, since in these cases there was time for extensive growth prior to death.

7. *Histology.* There have been a few recent attempts to correlate survival rates with subdivisions of the group of glioblastoma multiforme[2] as devised by Bailey and Cushing.[1] In our experience these subdivisions are difficult to use. The subtypes overlap, both in the same case and in different cases. The multiform aspect of the glioblastoma is such that if a large number of sections be taken, all varieties of cellular and vascular changes may be found.

SUMMARY AND CONCLUSIONS

In a series of 70 verified cases of glioblastoma multiforme, a greater proportion of males to females was found in the ratio of 1.8:1. Five patients survived 6 years or longer from the time of the first symptom, the longest being 14 years. An analysis of factors in survival revealed that operation had no significant effect on longevity; irradiation a slight effect, if any. The longest survivals occurred in patients with onset at from 24 to 42 years of age. However, age at onset was not a constant prognostic factor. The sex of the patient, the location of the neoplasm, or the histologic appearance gave no indication as to longevity. A significant lengthening of survival was found in those patients whose initial symptom was a focal motor seizure. While this was generally true, some patients with this symptom had a short survival. At the present time, there is no means, clinical or histologic, of predicting in a given case which patient will survive longer than the average.

The possibility is considered that in certain cases, an originally benign neoplasm becomes malignant and is seen as a glioblastoma multiforme at autopsy.

It is suggested that in reporting survival in cases of neoplasms of the nervous system, the survival from the first symptom as well as the postoperative survival be given.

Mr. Antol Herskovitz prepared the photomicrographs.

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MUSCLE WEAKNESS AND WASTING IN SCIATICA DUE TO FOURTH LUMBAR OR LUMBO-SACRAL DISC HERNIATIONS*

ERIC KUGELBERG, M.D., AND INGEMAR PETERSÉN
Department of Clinical Neurophysiology, Serafimerlasarettet, Stockholm, Sweden

(Received for publication November 2, 1949)

ISOLATED observations regarding the occurrence of paresis of dorsiflexion of either the great toe or the foot, or of both, as an indication of herniation of the 4th lumbar disc, were first reported by several authors.[12,13,14,15] The fact that definite diagnostic conclusions regarding the level of the herniation could be drawn from the distribution of the paresis was shown in extensive studies by McKenzie and Botterell,[9] and Norlén.[11] The first-mentioned authors attributed weakness of the dorsiflexors of the ankle to a lesion of the 5th lumbar root (4th disc), and of the plantar flexors to a lesion of the 1st sacral root (5th disc). Norlén further noted that among the dorsiflexors of the ankle and toes, that of the great toe was most commonly involved, being affected in 25 per cent of 56 cases with herniation of the 4th lumbar disc. In 43 patients with lumbosacral disc herniation, however, there was no weakness of dorsiflexion of the great toe.

Bradford and Spurling[1] summarize the current conception thus—the 5th lumbar root, which is injured by the 4th lumbar disc herniation, is the lowest important root supplying the dorsiflexors of the foot and toes, and the 1st sacral root, which is injured by the lumbosacral disc herniation, is the highest important root supplying the plantar flexors. Hitherto only the long extensors and flexors of the foot and toes have been studied, while the short toe extensors and the remaining small muscles of the foot have been disregarded.

As the long motor nerve fibers to the peripheral part of the extremities are more easily injured by various agents, e.g. ischemia, than the short fibers to the proximal parts,[5,8] we have examined the small muscles of the foot in patients with sciatica. It was found that the short extensor of the toes is far more frequently involved in 4th lumbar disc herniations than the long dorsiflexors, but is rarely affected in lumbosacral disc herniations. The condition of this muscle is therefore of considerable importance in determining the level of the herniated disc, as preliminarily reported.[7]

METHOD AND TECHNIC

Material. The material consisted of 66 patients with sciatica admitted to the surgical wards for operation. There were 44 men, and 22 women. The oldest was 65 years of age, and the youngest 21, with the average age over 40. The duration of the symptoms varied from 1 month to 10 years.

* This study was aided by a grant from the Swedish Medical Research Council.

In 41 patients the disc herniation occurred between the 4th and 5th lumbar vertebrae, and in 25, between the 5th lumbar and the 1st sacral vertebrae. In every patient operation confirmed the diagnosis of herniated disc.

Technic. Each patient was carefully examined clinically by both authors in regard to weakness which, in the majority of cases, was also analysed electromyographically. In both these examinations the patient was required voluntarily to contract the muscles to the maximum. In patients with incapacitating pain the greatest care was taken to ascertain whether a true paresis existed, or whether there was merely a functional reduction of strength due to pain. Here the electromyogram has been a valuable control, because the frequency of the individual action potentials is a good index of the degree of contraction.

In electromyography the current method of evaluation has been followed.[6] The muscles of both sides have always been compared, and only those with clear neurogenic changes have been regarded as abnormal.

In the clinical examinations only a definite weakness, compared with the same muscle on the other side, has been counted. It is difficult clinically to confirm paresis in the short flexors and extensors of the toes, owing to the dominating strength of the long muscles. We therefore had to examine the short muscles for atrophy and softness by palpation, with the patient maximally flexing the toes against resistance. The short toe extensors vary in size, but in normal cases neither asymmetry nor pathological electromyographical pictures have been observed. In a few cases the muscle was difficult to examine because of edema or fat, and in these patients the state of the muscle could only be evaluated by electromyography.

The short toe flexors are considerably more difficult to evaluate clinically and electromyographically than the short toe extensors. Therefore no definite conclusions can be drawn from examinations of the flexor muscles.

RESULTS

4th Lumbar Disc Herniation—41 cases (Table 1)

Extensors of Foot and Toes. Some form of motor disturbance—atrophy, paresis, or the electromyographical picture of a peripheral motor neuron lesion—was found in 37 cases. The extensor digitorum brevis was the muscle most often affected(Fig.1). In no less than 35 patients either atrophy was clearly visible (Fig. 2) on the affected side (23 cases), or there was, on maximal contraction, a palpably softer consistency compared with the corresponding muscle of the other foot (12 cases). In 35 cases there was also electromyographical evidence of a lesion of the lower motor neuron.

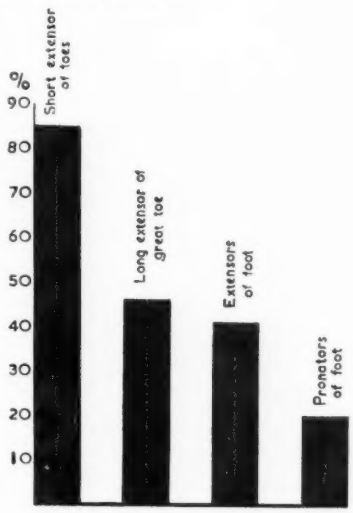

FIG. 1. Chart showing distribution of motor disturbances within the extensor group.

The electromyogram gave, with one or two exceptions, the same results as the clinical tests, thus proving a good control.

There was weakness of dorsiflexion of the great toe in 19 cases, of dorsiflexion of the small toes in 12 cases, of dorsiflexion of the foot in 17 cases, and of pronation of the foot in 8 cases (Fig. 1). The electromyographical

FIG. 2. Fourth lumbar disc herniation (Case 35). The only clinical sign was extreme atrophy of M. extensor digitorum brevis. (A) Affected foot. (B) Normal foot for comparison.

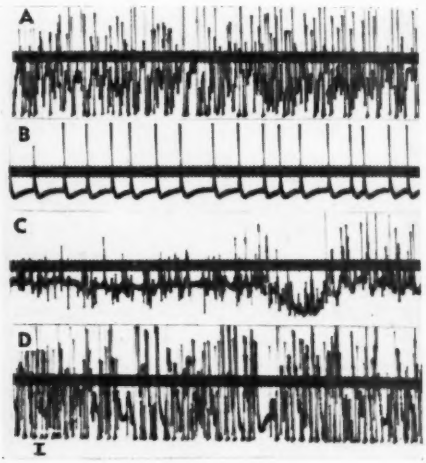

FIG. 3. Fourth lumbar disc herniation. Electromyographic recordings of maximal voluntary contractions from same patient as in Fig. 2. (A) Extensor digitorum brevis on normal side. (B) Same muscle on affected side, showing marked diminution of motor unit activity. (C) Extensor hallucis longus on affected side, showing approximately normal motor unit activity. (D) Tibialis anticus on affected side, showing normal motor unit activity. Time: 1/100 sec. Calibration: 100μV.

control of the *M. extensor hallucis longus*, *M. tibialis anterior* and *M. peroneus longus* corresponded in the majority of cases to the clinical findings. If there were electromyographical changes in several muscles at the same time they were nearly always more pronounced in the short extensors of the toes (Fig. 3).

Motor disturbances are, as already mentioned, most common in the short toe extensors. In 2 patients, however, the M. extensor digitorum brevis showed normal clinical findings at the same time as there was paresis of one or two long extensors. In both these cases, however, the electromyographical picture was pathological. The muscles of the non-operated side were normal, except in a few patients who had previously had alternating right– and left-sided pain.

TABLE 1

Fourth Lumbar Disc Herniation

Case No.	M. ext. dig. brev.	Dorsal Flexion of Great Toe	Dorsal Flexion of Foot	Dorsal Flexion of Toes	Pronation of Foot	Plantar Flexion of Foot & Toes	Achilles Reflex
1	Normal	Normal	Normal	Normal	Normal	Normal	Absent
2	"	"	"	"	"	"	"
3*	"	"	"	"	"	"	"
4	"	Paresis	"	Paresis	"	Paresis (toes)	Normal
5	"	Normal	"	Normal	"	Normal	"
6	"	Paresis	"	"	"	Paresis (foot)	"
7	Reduced musc. consistency	Normal	"	"	"	Normal	Absent
8	"	"	"	"	"	"	Weakened
9	"	"	"	"	"	"	Normal
10	"	"	"	"	"	"	"
11	"	Paresis	Paresis	Paresis	"	"	"
12	"	Normal	Normal	Normal	"	"	"
13	"	"	"	"	"	"	"
14	"	Paresis	"	"	"	"	"
15	"	"	"	"	"	"	"
16	"	"	Paresis	"	"	"	"
17	"	"	"	Paresis	"	"	"
18	"	"	"	"	Paresis	Paresis (foot)	Weakened
19	Atrophy	Normal	"	Normal	Normal	Normal	Normal
20	"	Paresis	"	"	"	"	"
21	"	"	"	Paresis	Paresis	"	Absent
22	"	Normal	Normal	Normal	Normal	"	Normal
23	"	"	"	"	"	"	"
24	"	"	"	"	"	"	"
25	"	"	"	"	"	"	"
26	"	"	"	"	"	"	Weakened
27	"	Paresis	"	Paresis	"	"	Normal
28	"	"	Paresis	Normal	Paresis	"	"
29	"	"	"	Paresis	"	"	"
30	"	"	"	"	"	"	"
31†	"	Normal	"	Normal	Normal	Paresis (foot)	Absent
32	"	"	"	"	"	"	"
33	"	"	Normal	"	"	Normal	Normal
34	"	Paresis	"	"	"	"	"
35	"	Normal	"	"	"	"	"
36	"	"	"	Paresis	"	"	"
37	"	"	Paresis	Normal	"	"	"
38	"	Paresis	"	"	"	"	"
39	"	"	"	Paresis	Paresis	"	"
40	"	"	"	"	"	"	"
41	"	"	"	"	"	"	Absent

* Sensory root section of S1 performed earlier.

† L5 and S1 roots compressed.

Flexors of the Foot and Toes. In 5 cases there was paresis of the plantar flexors of the foot and toes. In 2 of these compression of L5 as well as S1 roots was found at operation. In all cases of flexor paresis there were also disturbances of the extensor group (Fig. 4).

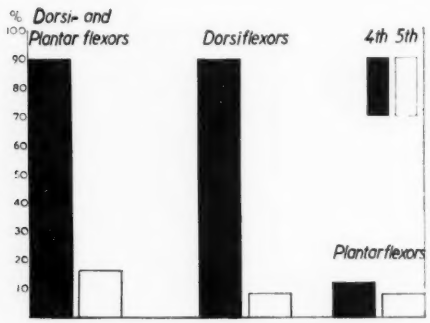

FIG. 4. Distribution of clinical motor disturbances in 4th (41 cases) and 5th (25 cases) lumbar disc herniation.

Achilles Reflex. This reflex was normal in 31 cases, weakened in 3, and absent in 7. In 1 case with absent ankle jerk, rhizotomy of the S1 sensory nerve root had been performed earlier. Our results regarding the absence of the Achilles reflex in the L5 syndrome corresponded to reports from other authors,[11,16] although we did not find diminution of the Achilles reflexes so frequently.

TABLE 2

Lumbosacral Disc Herniations

Case No.	Atrophy of M. ext. dig. brev.	Paresis of Extensors of Foot and Toes	Paresis of Flexors of Foot and Toes	Achilles Reflex
1	0	0	0	Normal
2	0	0	0	Weakened
3	0	0	0	Absent
4	0	0	Great toe	"
5	0	0	0	"
6	0	0	Foot and great toe	"
7	0	0	0	"
8	0	0	0	
9	Slight	All toes	0	Normal
10	0	0	0	Absent
11	0	0	0	Weakened
12	0	0	0	Absent
13	0	0	0	Normal
14	0	0	0	Weakened
15	0	0	0	"
16	0	0	0	"
17	0	0	0	Absent
18	0	0	0	"
19	0	0	0	"
20*	0	0	0	Normal
21	0	0	0	Weakened
22	0	0	0	"
23	0	0	0	Absent
24	Slight	0	0	Weakened
25	0	0	0	Absent

* The roots below S1 were compressed.

Lumbosacral Disc Herniations—25 cases (Table 2)

Motor Disorders. In 21 of the 25 patients there was no clinical weakness or wasting of the flexors or extensors of the foot and toes. Of the remaining 4 patients, 2 had weakness of the long plantar flexors, and 2 had slight wasting of the *extensor digitorum brevis*, in 1 case combined with weakness of the dorsiflexors of all toes.

Compared with the 4th lumbar disc herniations, the motor changes in lumbosacral disc herniations are strikingly sparse (Fig. 4).

The Achilles Reflex. This was absent in 13 cases, weakened in 8 cases, and normal in 4, corresponding well with the findings of earlier authors.[11,16]

DISCUSSION

In our material only those patients have been included in whom the diagnosis of herniated disc was confirmed at operation. The results cannot therefore be directly applied, for example, to disc herniation material from an outpatient department where a greater number of mild cases are seen.

The clinical diagnosis of the level of the disc herniation can, of course, be carried only to a certain point. Definite diagnostic signs and symptoms may be completely absent, or the prolapsed disc may compress several roots at the same time (No. 31, Table 1); a root other than that expected may be compressed (No. 20, Table 2); or the symptoms may be atypical, possibly because of deviations from the normal in the anatomical composition of the root (No. 9, Table 2). These are examples from our material of factors that limit the clinical possibilities.

Usually, however, it is possible to determine the level clinically. The motor symptoms are important here, and the principal object of this study is to show the remarkably common affection of the *M. extensor digitorum brevis* in 4th lumbar disc herniations. In fact, it appears to be the most constant sign of disc herniation at this level. No detailed study on the distribution of the sensory disturbances has been made in our work, which is primarily directed towards motor disorders. However, judging from the literature, radiating pain or impairment of sensibility, with distribution referable to a definite single root, do not occur in such a large percentage of cases. Nevertheless, it is clear that all the signs and symptoms must be collected and evaluated in each patient.

The examinations further show that the short extensor of the toes has its main nerve supply from roots above S1, and that it is not as a rule affected in lumbosacral disc herniations. In 1 case with an S1 root syndrome exploration revealed a neurinoma, and the nerve had to be divided. In spite of this there were no clinical or electromyographical signs of involvement of the short extensor. Conversely, there are many cases in our material where the short extensor of the toes has been extremely wasted without any electrical activity on attempted voluntary contraction. Exploration revealed only compression of the 5th lumbar root, indicating that the main nervous supply of the short extensor came from L5. Our findings do not support the opinion

that the muscle is innervated from S1 and S2 (Haymaker and Woodhall,[3] and others[10]), but compare better with most textbooks, which suggest L4, L5, and S1.

The more constant involvement of the extensor brevis, compared with the long dorsiflexors of the foot and toes, is not due to the examination technic, since the electromyogram clearly shows that the short extensors are affected more than the long extensors. This difference may be due to less segmental overlap in the innervation of the muscles in the distal part of the extremity than in the proximal, as is assumed for sensory innervation (Keegan,[4] Falconer *et al.*[2]). Another possibility is the greater vulnerability of the long nerve fibers compared with the shorter ones, as found, for instance, in experimental ischemia on the human arm (Lewis, Pickering and Rothschild,[8] and Kugelberg.[5]). In fact, if the circulation to the leg is arrested by compression with a pneumatic cuff, within the extensor group the short extensor is paralysed first, followed in order by the *extensor hallucis longus*, the *extensor digitorum longus*, the *tibialis anterior*, and the *peroneus longus*.

SUMMARY

Sixty-six patients with sciatica due to 4th lumbar (41 cases) or lumbosacral (25 cases) disc herniation verified by operation, have been examined for muscular disturbances in the lower leg and foot.

1. *4th Lumbar Disc Herniation.* In 90 per cent of the cases the muscles within the extensor group were affected with the following distribution: *M. extensor digitorum brevis* in 85 per cent (atrophy or a palpably soft consistency on maximal contraction); *M. extensor hallucis longus* in 46 per cent (weakness); *M. tibialis anterior* in 41 per cent (weakness).

In 13 per cent of the cases there was weakness of the muscles of the flexor group. In these cases there were also disturbances of the extensor group.

2. *Lumbosacral Disc Herniation.* In only 16 per cent of the cases were the muscles affected, with weakness and wasting of those of the extensor group in 8 per cent, and of the flexor group in 8 per cent.

3. The cause of the strikingly common affection of the *M. extensor digitorum brevis* in 4th lumbar disc herniation is discussed.

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CASE REPORTS AND TECHNICAL NOTES

THE CONTROL OF ACUTE SUBDURAL HEMORRHAGE BY INJECTION OF SALINE INTO THE SPINAL SUBARACHNOID SPACE

ROSS S. MCELWEE, M.D., AND BRONSON S. RAY, M.D.
Department of Surgery, New York Hospital—Cornell Medical College, New York, New York

(Received for publication August 10, 1949)

The case to be reported is unique in two respects: (a) after a relatively simple head injury subdural bleeding occurred in such degree that it threatened the patient's life within a few hours; and (b) the bleeding was controlled by the tamponade effect of expansion of the brain through injection of saline into the spinal subarachnoid space.

Although bleeding into the subdural space may occur with any type of injury to the head the amount is usually relatively small and it is not commonly expected that important signs of its presence will occur for at least several days. Kennedy and Wortis[2] in 1936 made a study of "acute" subdural hematoma and made a plea for more frequent use of early trephining in patients suspected of having the lesion. But this concept has not had wide popularity because the evacuation of blood from the subdural space during the early and critical period after a head injury seldom alters the situation favorably. Browder[1] in a critical review of 289 cases of subdural hematoma found a mortality of 82 per cent in patients operated upon in the first 24 hours after injury and concluded that "the removal of hematomas during the first twenty-four hours . . . is seldom justifiable."

The problem of obliterating the space occupied by a hematoma after its evacuation has been of some concern in the treatment of chronic subdural hematomas. Common methods employed in the past for expanding the brain have been the use of intravenous isotonic or hypotonic fluids, lowering of the head, compression of the jugular veins, and intraventricular injection of fluid. LaLonde and Gardner[3] in 1948 reported good results in expansion of the compressed cerebral hemisphere by spinal injection of physiologic saline solution.

As far as is known the intraspinal injection of saline has not been previously described as a method of controlling acute subdural bleeding. Its effect was so successful in this case that it was thought desirable to bring the experience to the attention of others who might be confronted with a similar emergency.

NYH #503125. J. D., a 33-year-old lawyer, was first admitted to the New York Hospital in 1947 with a history of occasional generalized convulsions for the previous 2 years. At that time general physical and neurological examinations were unremarkable. The CSF was normal, the EEG was normal, and the pneumoencephalogram gave evidence of slight cerebral atrophy. On discharge from the hospital he was advised to take 30 mg. of phenobarbital 3 times a day. He followed instructions poorly and frequently omitted his medication for several days at a time. He had one brief convulsive episode 2 months before admission.

On the night of his 2nd admission, June 19, 1949, he had a generalized convulsion and fell forward striking his face against a radiator. His left eye was bruised and one lip was cut. He was unconscious for about 5 minutes and on awakening complained only of generalized headache. Two hours later his headache became extreme, he lapsed into coma and was brought at once to the hospital.

Examination. He was wholly unresponsive and his condition appeared extremely serious. Breathing was stertorous and rapid. B.P. was 100/90, pulse 95 and temperature normal. The right pupil was widely dilated. The extremities were held rigidly in extension and the deep tendon reflexes were hyperactive. There was a bilateral Babinski sign. X-ray of the skull did not reveal a fracture.

Operation. A lumbar puncture revealed clear CSF with a pressure of 180 mm. of water. A right temporoparietal trephine was made, revealing a tense bluish dura. When the dura was incised, unclotted dark blood gushed forth almost as fast as it could be sucked away. The cortex could be seen depressed about 2 cm. from the dura by the large quantity of blood. The blood appeared to be venous and it was soon evident that active bleeding was continuing, but through the small opening there was no possibility of locating and controlling the torn vessel. It seemed likely that lowering the head or the injection of intravenous fluids would only add to the venous bleeding. As the blood loss continued, the patient's condition rapidly deteriorated and his blood pressure could not be obtained. At this point he was turned on his left side and a lumbar puncture performed. About 40 cc. of saline were injected in 3 or 4 minutes. During this time the brain steadily expanded to obliterate the subdural space and the hemorrhage ceased. The wound was closed without drainage after a few minutes' observation.

Course. The patient's condition improved quickly and he began to regain consciousness within 12 hours. By the 2nd day it was apparent that he had a right 3rd cranial nerve paralysis and a right hemiparesis without aphasia, sensory changes or hemianopsia. The left side of the brain was then explored through a temporoparietal trephine disclosing a small amount of clear fluid in the subdural space. The right wound was reopened but only a small collection of thin bloody fluid was encountered.

FIG. 1. Photograph of patient about 3 weeks after the accident showing unresolved oculomotor paralysis and facial weakness (part of the hemiparesis) on the side of the subdural hemorrhage.

The patient improved for several days and then his condition deteriorated. Daily spinal taps revealed elevated pressures and the reduction of this pressure to normal seemed to have a beneficial effect. Intravenous fluids and feedings by gavage were necessary for 14 days and then discontinued as he regained ability to swallow. The right 3rd nerve paralysis and the right hemiparesis gradually improved during his hospitalization (Fig. 1) and he was able to care for himself on his discharge from the hospital 30 days after operation. A month later he had improved greatly and had only a slight right-sided weakness.

SUMMARY AND CONCLUSION

During a generalized convulsive attack this man sustained an injury to the front of the head. A short lucid period was followed by coma and his condition rapidly became serious. A lumbar tap revealed clear fluid under normal pressure. In the absence of other localizing signs the exploratory burr hole was made on the side of the dilated pupil. Active subdural venous hemorrhage was encountered. In all probability the bleeding was from one of the cerebral veins which cross the subdural space and was torn by the force of the head injury. There was no possibility of locating the torn vessel but the tamponade effect of the brain expanded by the

intraspinal injection of saline proved adequate. That the brain remained expanded and the hemorrhage controlled were demonstrated by re-exploration 2 days later. The homolateral hemiparesis in all probability was the result of compression of the cerebral peduncle against the incisura of the tentorium and bears witness to the degree of the hemorrhage and the resultant shift of the brain.

The intraspinal injection of saline to expand the brain and obliterate the subdural space may be a life-saving measure in the control of acute subdural hemorrhage.

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PROFUSE SUBARACHNOID HEMORRHAGE CAUSED BY CEREBRAL GLIOMA

Dean H. Echols, M.D., and Frederick C. Rehfeldt, M.D.
Department of Surgery, Tulane University of Louisiana School of Medicine, and Section on Neurosurgery, Ochsner Clinic, New Orleans, Louisiana

(Received for publication August 23, 1949)

Gliomas are rarely provocative of gross subarachnoid hemorrhage. In 1937 Russel and Kershman[3] reported 1 case of their own and referred to another by Strauss, Globus and Ginsburg.[4] Hesser,[2] who reviewed the literature in 1948, was able to find no additional cases but added 1 of his own. In these 3 cases the spinal fluid was grossly bloody at some time during the course of the disease. Russel and Kershman's patient had only 430 red blood cells per c.mm. in the spinal fluid on admission but after a few days in the hospital experienced a severe exacerbation at which time the spinal fluid became grossly bloody; a glioblastoma multiforme was demonstrated at necropsy. In Hesser's case a spinal puncture done by another physician shortly after rather sudden onset of severe head pain revealed grossly bloody fluid but when the patient was examined by Hesser several weeks later, only 3 red blood cells per c.mm. were demonstrated in the spinal fluid; the diagnosis at necropsy was glioblastoma multiforme. Strauss, Globus and Ginsburg[4] reported their case as 1 of 150 gliomas; a single spinal puncture before operation produced blood-stained fluid. Thus, the case which forms the basis of this report, that of a verified glioblastoma multiforme which produced persistently bloody spinal fluid, presenting a clinical picture of leaking aneurysm or angiomatous anomaly, would seem to be the fourth to be recorded in the American medical literature.

REPORT OF CASE

J. T. F., a 42-year-old white man, sought medical assistance when headaches which he had experienced intermittently for 8 months, became so severe that he was unable to work. The

pain, which involved the left side of the head, varied in duration and intensity and was frequently accompanied by nausea and vomiting. After finding the CSF to be grossly bloody on 6 occasions, his physician made a diagnosis of leaking intracranial aneurysm, and referred the patient for surgical treatment. On admission to Touro Infirmary on Dec. 7, 1942, 9 months after onset of the illness, he was disoriented but able to give the additional history that he also had failing vision and some loss of hearing.

Examination revealed choked disks, nuchal rigidity, facial weakness of central type on the left side, weakness of the legs, absent knee and ankle jerks, and normal plantar reflexes. The CSF, which was bloody and xanthochromic, was under a pressure of 420 mm. in the water manometer.

A pneumoencephalogram, made Dec. 9, 1942, revealed slight elevation of the frontal horn of the right lateral ventricle and questionable shift of the ventricular system to the left. As it was believed that this deformity was produced by a hematoma from a leaking aneurysm, an angiogram was made the following day.

Treatment and Course. Although the arteriogram was normal, the right internal carotid artery was ligated in the neck.

The amount of blood in the CSF diminished for several days. However, on Dec. 17, 1942, when more bleeding occurred, it was decided to approach the "aneurysm" directly.

1st Operation. On Dec. 19, 1942, as the circle of Willis was being exposed through a right frontal craniotomy, a reddish, friable, racemose tumor, 2 cm. in diameter, was found at the tip of the frontal lobe. Although no blood clots could be seen in the vicinity, this lesion was obviously the source of the bleeding. It was adherent to the dura but easily separated from it. The lesion and a small portion of brain were removed.

The histologic diagnosis was astroblastoma.

Postoperative Course. The CSF became clear for the first time in 2 months and on the 17th postoperative day the patient was well enough to be discharged. He was able to work as a carpenter for 8 months before he was forced to stop because of weakness of the legs.

Readmission. He returned on Feb. 13, 1944, 14 months after operation, too ataxic to walk or feed himself. He had been experiencing severe headaches accompanied by vomiting for a few days. For the last 2 days he had had urinary and fecal incontinence. Content and stream of talk were deteriorated; he could, however, comprehend simple instructions. Spinal puncture was not attempted.

2nd Operation. On Feb. 15, 1944, the right frontal flap was reopened under local anesthesia. The brain herniated when the dura was opened. A large, smooth-walled cyst containing yellow fluid occupied the anterior half of the right frontal lobe. The cyst and much of the adjacent neoplastic tissue were removed.

Microscopically, the tumor was considerably more malignant than at the time of the first operation and the diagnosis was advanced to glioblastoma multiforme.

Course. It has not been possible to follow the patient since his last discharge from the hospital but death within a month or two was anticipated.

COMMENT

It certainly seemed plausible to suspect that a patient in the fifth decade of life, with a history of headache, grossly bloody spinal fluid and signs of increased intracranial pressure had a leaking aneurysm with the formation of a hematoma. The encephalographic observations of elevation of the frontal horn of the right lateral ventricle as well as slight ventricular shift to the left tended to corroborate the presence of hematoma.A normal angiogram is still not considered to be proof of the absence of an aneurysm. However, pain in the head associated with ruptured aneurysm tends to be sudden and severe, and in this case it became progressively more severe, which is more characteristic of brain tumor. We were aware that red blood cells may appear in microscopic amounts[1] in the spinal fluid in patients with brain tumor, but such a striking and persistent hemorrhage was a new experience.

The fact that the spinal fluid became clear postoperatively and remained so would appear to be strongly indicative that the tumor was responsible for the hemorrhage.

SUMMARY

A case is presented of subarachnoid hemorrhage with grossly bloody spinal fluid provoked by a verified glioblastoma multiforme, simulating ruptured aneurysm with hematoma. It is believed to be the fourth to be reported in the American medical literature.

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ARNOLD-CHIARI MALFORMATION

Everett F. Hurteau, M.D.*
Akron, Ohio

(Received for publication October 10, 1949)

It has repeatedly been mentioned that Arnold-Chiari malformation without abnormality of the cervical spine is unusual,[1,2] and the difficulties in diagnosing the lesion under such circumstances have been indicated. The following case is thus presented for its statistical value and may contribute something of diagnostic and therapeutic value as well.

CASE REPORT

A 23-year-old white female was admitted to the hospital on Feb. 22, 1948 complaining of headache which began about 1 year previously. This symptom had been progressive and accompanied by vomiting during the last month. Falling to the left when walking, dysphagia and dysarthria had been noted for the last 3 months. Tinnitus, progressive impairment of hearing on the left and progressive impairment of vision had been present for 1 month.

Examination revealed an apprehensive and emaciated patient complaining bitterly of headache. Her weight was only 76 pounds. Vision was impaired to such an extent that she retained only ability to count fingers. There were 3 D. of choked disc with retinal hemorrhages and exudates. Signs of 5th cranial nerve involvement on the left were evident, with impaired corneal sensation and hypoalgesia over the distribution of the 1st and 2nd divisions. There was subjective diplopia on looking to the left without objective strabismus, and minimal impairment of ability to wrinkle the left side of the forehead. Air conduction and bone conduction were diminished on the left but vestibular tests reportedly showed hyperirritabil-

* 175 S. Main Street, Akron 8, Ohio.

ity of the left labyrinth. Dysarthria and dysphagia of mild degree were present without diminution of gag reflex. Cerebellar signs were confined to the left side and consisted of intention tremor, inaccuracy of the finger-to-nose test, positive Holmes rebound sign, and diminished deep reflexes in the upper and lower extremities.

Spinal puncture showed a pressure of 240 mm. of water, 2 cells and a total protein of 14 mg. per cent.

X-rays of the cervical spine were normal. X-rays of the skull showed some flattening of the squama of the occipital bone and a resultant shallow posterior fossa (Fig. 1). There was no evi-

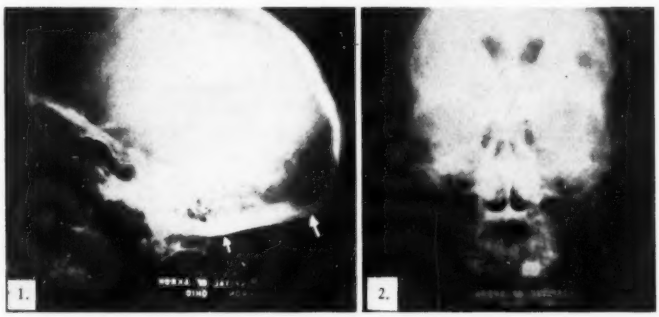

FIG. 1. Preoperative X-ray showing loss of convexity of the squama of the occipital bone (between arrows) and resultant shallowness of the posterior fossa.

FIG. 2. Ventriculogram showing lack of hydrocephalus.

dence of platybasia. Ventriculography on Feb. 27, 1948 (Fig. 2) showed no hydrocephalus. In retrospect, it was noted that the 4th ventricle was nearer the foramen magnum than usual.

A *suboccipital craniotomy* was performed on Mar. 2, 1948, using the inverted U type of incision with the patient in the sitting position. Careful exploration of the cerebellopontine angles and 4th ventricle revealed no neoplasm. There was, however, a very striking Arnold-Chiari malformation. The tips of the cerebellar tonsils were near the level of the upper margin of the 3rd cervical arch and attached to the brain stem and upper cervical cord by adhesions. The caudal six cranial nerves followed an unusually oblique rostral course from their point of emergence from the brain stem to enter their foramina at an increased angle.

The cerebellum was carefully freed and CSF was observed to flow into the wound from the 4th ventricle. The dura was left widely open over the suboccipital and upper cervical regions. At the completion of these procedures there was a distance of about 5 mm. between the lower margin of the dural opening and the caudal margin of the cerebellar tonsils.

Course. She was discharged from the hospital on Mar. 25, 1948. At this time she was free of headache, her papilledema was receding and visual acuity was approaching normal.

On Apr. 19, 1948 she again experienced headache, and examination revealed bulging and tenseness of the suboccipital wound. During the ensuing 7 months there were remissions and exacerbations of signs and symptoms of increased intracranial pressure. The ataxia and cranial nerve palsies did not recur. Aspiration of CSF from the suboccipital wound repeatedly relieved her headache temporarily.

A third ventriculostomy was done on Nov. 27, 1948. The right transtemporal route was used and an opening was made in the lateral wall of the 3rd ventricle, just anterior to the 3rd cranial nerve. Indigo carmine injected into the right lateral ventricle promptly escaped from the ventriculostomy opening. Following this procedure headache and bulging of the suboccipital wound improved for 5 days, then recurred.

On Dec. 8, 1948 indigo carmine injected through the suboccipital scalp into the pseudomeningocele appeared in the lumbar subarachnoid space only after 21 minutes, and even then

only in very small amounts. CSF withdrawn prior to these injections contained 173 mg. per cent of protein.

Re-exploration of the suboccipital area was performed on Dec. 11, 1948, this time through a midline incision and with the patient in a prone position. The cerebellar hemispheres were found to be firmly attached to the margin of the dural opening. As a result the CSF upon leaving the 4th ventricle was prevented from entering the basal cisterns. The lamina of the 3rd and 4th cervical vertebrae were removed and the dura was opened as far caudally as possible, thus creating a considerable space between the tip of the cerebellar tonsils and the margin of the dura. This accomplished a good communication between the spinal canal, the 4th ventricle and the basal cisterns. Indigo carmine injected into the lateral ventricle promptly appeared in the wound. Phenolsulphonphthalein, injected into the lumbar theca, also appeared promptly. The dura was left widely open and the remainder of the wound carefully closed.

Course. The patient made an uneventful recovery. When last seen on Sept. 30, 1949 she had gained 38 pounds. The suboccipital wound was soft. She had experienced no recurrence of headache. Her optic discs were normal. Other cranial nerves showed no sign of involvement. There was no subjective or objective evidence of ataxia.

DISCUSSION

There were several unusual features in this case. Loss of convexity of the occipital bone has not, to the author's knowledge, heretofore been reported in conjunction with Arnold-Chiari deformity. This may be a coincidental finding, but seems worthy of mention. Papilledema was quite marked and was not accompanied by the hydrocephalus one would anticipate with a block in the posterior fossa. The failure of the first operation was undoubtedly due to an inadequate laminectomy. This mistake was a result of the effect of extreme flexion of the head on the relative position of the 3rd cervical arch and the lower margin of the cerebellum. With the head in this position there were several mm. between the lower margin of the dural opening and the cerebellum, and the illusion of an adequate laminectomy was thus created. After the wound was closed, however, and the head brought back to the neutral position, the tips of the cerebellar tonsils presumably were brought into contact with the dural edge. During the course of wound repair adhesions between these surfaces ensued and resulted in recurrence of the CSF block. Third ventriculostomy failed, probably due to closure of the stoma. The second and more complete laminectomy has, it is hoped, permanently re-established communication of the CSF pathways.

SUMMARY

A case of Arnold-Chiari malformation without malformation of the cervical spine is presented. Several unique findings, including a malformation of the basi-occiput are mentioned. The danger of failure of operation due to inadequate laminectomy is discussed.

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AN AUTOMATIC DRILL FOR CRANIOTOMY*

GEORGE W. SMITH, M.D.
Department of Neurological Surgery, University of Maryland, Baltimore, Maryland

(Received for publication September 15, 1949)

A combined perforator and burr has been designed which makes a sheer, complete trephine opening requiring only one instrument and which will automatically stop drilling by allowing the driving force to be disengaged once the last shelf of inner table is removed at the base of the opening. This instrument, which can be driven either manually or by motor, makes a trephine opening *speedily* and safely with no injury to meninges or brain.

The drill is made of chrome-plated high-speed steel and weighs less than 3 oz. It is 2½" long and the center bore is 9/16" in diameter, with the counter bore being 11/16" in diameter. The counter bore is 3/16" shorter than the center bore. The cutting surfaces of the center bore are approximately 18° positive pitch with 4 straight flutes. The counter bore has the same pitch without any undercutting. The shank is interchangeable so as to fit any chuck. Fig. 1 shows the drill in a side view, disengaged. Fig. 2 shows an end view of the 4 cutting surfaces. These cutting surfaces are similar to those of a perforator-drill described by Carmody.[1] Fig. 3 shows the drill in the engaged state.

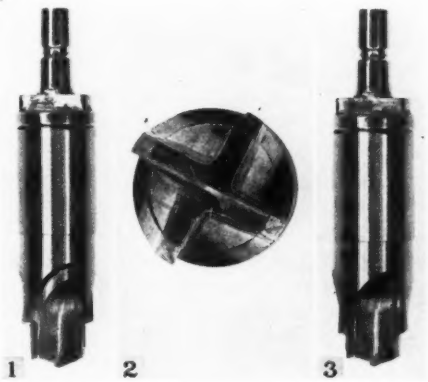

FIG. 1. Side view with drill in disengaged state. Note added length of center bore. This is the only difference between Fig. 1 and Fig. 3.

FIG. 2. End view of drill, showing the 4 flutes and cutting surfaces. Note counter bore, which appears as an outer collar. The counter bore works in synchrony with the center bore and has cutting surfaces.

FIG. 3. Side view of perforator in engaged state. Note: upper driving segment that fits into the chuck; counter bore (shorter than center bore); center bore and point.

The drill is made in 3 functioning parts: the 1st an upper driving part that fits into the chuck of the drill; the 2nd part is the center bore, which has the 3rd part surrounding it as a collar—this is the counter bore. The two lower parts, center point and counter bore, are locked

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* Exhibited before the Society of Neurological Surgeons, Baltimore, Maryland, June 10, 1949.

Financial assistance for this project was given by the Hoffberger Neurosurgical Fund, University Hospital, Baltimore, Maryland.

in the rotary plane at all times by a key, but move freely upon one another in the vertical plane. These two lower segments then rotate in synchrony. When the center bore or point is depressed 1/16″ it pushes the key into position and locks the lower units with the 3rd and driving segment (the upper portion).

The engagement and functioning of the drill are dependent upon pressure being maintained on the center bore by an intact skull.* Once the outer and inner tables of the skull are penetrated, regardless of their thickness, the pressure on the center bore is no longer maintained and this is free to advance 1/16″ only and in thus doing automatically disengages the driving force of the bore and counter bore cutting surfaces. The counter bore, which is $\frac{1}{8}$″ *larger in diameter* and trailing the center bore by 3/16″ in depth obviously supports the drill and weight of the operator by not being able to advance farther into the completely cut opening. The functioning of the disengagement is dependent upon the 60° pitch of the locking key and the center bore being under slight spring tension, thus "walking" the engagement key out of its engagement shoulder once pressure is no longer encountered by the center drill or bore.

The caliber of the center bore or drill is uniform in its entirety. Thus, when the drill has trephined the skull and automatically stops drilling, the opening is clean of bone chips, has sheer shoulders and is *completely* through the skull. The drill produces large generous bone chips which can be used to fill defects or be replaced in the trephine opening if desired.

This drill works equally well manually or by power. The optimum speed is approximately 250 rev./min. and should not be placed in a power-driven chuck that exceeds this. Any power-driven chuck whose speed is regulated by a rheostat and can be adequately controlled by the surgeon is satisfactory. The drill performs well in the Luck Saw motor chuck, which can be autoclaved.

This automatic releasing drill has been used with satisfaction at the University Hospital during the past 18 months.† In all cases it has made excellent trephine openings without any trace of injury to the meninges or brain.

COMMENT

A new method for trephining skulls offers the following advantages:

(1) A marked reduction in operating time.

(2) The safety factor is greatly increased.

(3) The variability in the thickness of skull is no longer a threat to the surgeon.

(4) The drill can be power-driven with safety.

(5) The drill produces *complete* adequate trephine openings with one instrument.

(6) Sizeable enough bone chips are produced that they may be replaced or used to fill a defect if desired.

(7) Only a minimum of energy is exerted by the surgeon.

Appreciation is extended to Mr. T. A. Jones of Baltimore, who fabricated the drill, and to members of the Neurological Surgery Staff of University Hospital for their assistance and trial of this drill.

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* In one case it was found not to properly engage. Closer inspection of the skull in this case showed markedly abnormal bone, being the site of a metastatic sarcoma. It is felt that this provides a method to determine the soundness of the skull.

† This instrument can be obtained from T. A. Jones, Mfg. Machinist, 837 E. Lombard Street, Baltimore, Maryland. The cost is $55.00.

NOTICES

AMERICAN NEUROLOGICAL ASSOCIATION

The next annual meeting of the American Neurological Association will be held in Atlantic City, New Jersey, on June 12, 13 and 14, 1950, with headquarters at the Claridge Hotel.

JOHN CRERAR LIBRARY

On March 20, 1950 announcement was made of Dr. Eric Oldberg's election to the Board of Directors of The John Crerar Library, Chicago, Illinois. His appointment fills a vacancy left by the resignation of Dr. Ludvig Hektoen, President of the Chicago Tumor Institute, and is in the tradition of a long line of leading professional and business men who have been responsible for the development of The John Crerar Library into the largest public library devoted exclusively to science, technology, and medicine. The Library was founded in 1894. Dr. Oldberg is the fifty-first man to join the distinguished line of directors, which has included such noted men as Marshall Field, Robert T. Lincoln, Ludvig Hektoen, Arthur H. Compton, and Andrew C. Ivy.

Dr. Oldberg is professor and head of the Department of Neurology and Neurological Surgery in the University of Illinois College of Medicine, a position he has held since 1936. He first joined the staff of the University in 1931. Previously he had been instructor in physiology at Northwestern University as Assistant Professor of Surgery, and had spent three years at the Peter Bent Brigham Hospital in Boston, first as Surgical House Officer, then as Assistant Resident Surgeon, and in 1930, as Resident Neurological Surgeon to Dr. Harvey Cushing. He is a fellow of the American College of Surgeons, and a member of numerous medical societies. He is an associate editor of the *Journal of Neuropathology.*

The John Crerar Library, in addition to its general scientific collections, has the largest medical collection in the Middle West, and ranks with the best medical libraries in the country outside the Army Medical Library. Its medical volumes alone exceed 130,000, and include good research collections in all of the specialties, those in ophthalmology, pediatrics, cancer, and cardiology being particularly strong due to special gifts in these fields.

NATIONAL PARAPLEGIA FOUNDATION

The National Paraplegia Foundation announces the establishment of a limited number of fellowships for research in spinal cord disease and trauma. These fellowships carry a minimum stipend of $3000 per year and may be awarded to any candidate who has demonstrated a capacity for medical research and has outlined a program of meritorious study. The fellowships will be awarded by the Medical Advisory Committee and are open for award for the academic year 1950–1951. Application forms may be obtained from Dr. L. W. Freeman, Chairman, 1040–1232 West Michigan St., Indianapolis 7, Indiana, and applications should be submitted to him not later than June 1, 1950.

NEUROSURGERY IS WHAT YOU MAKE IT*

FRANK TURNBULL, M.D.†
Vancouver, B. C., Canada

(Received for publication April 16, 1950)

AT THE BEGINNING

THE middle thirties was a stimulating period for neurosurgeons. The new specialty was about halfway through the years of expansion,[3] with an increasing demand for its services. This expansion was not yet apparent to the general medical profession. There was still some unbelief about the practicalness of such a restricted field. Because neurosurgery dealt chiefly with the brain its votaries appeared to move in a rather exotic atmosphere. Almost all of the members of the Harvey Cushing Society were professors, major or minor.

Into this galaxy two unorthodox members were introduced. They were starting to practice neurosurgery in far western communities that did not boast of medical schools. The entrance of Dr. Haven of Seattle and myself from Vancouver illustrates the dictum that there are no absolute truths. We never did hear the reason. We would like to think that it was because of the interest of Dr. Cushing himself, but he hardly knew us. When I first met Harvey Cushing in 1932, and again in 1935, he seldom saw me except in the company of Dr. Ken McKenzie. So I became, as I supposed happened to Hale Haven, what Dr. Cushing termed a "spiritual grandson."

During my early years in the Society I was always chagrined to admit that there was no medical school in Vancouver. One missed the atmosphere of the teaching center when away out West and always felt like a student again, when back. Later on, the large centers seemed to lose some of their glamour. Envy for opposite numbers changed to a wonder if they would not like to trade places. There came a day when one enjoyed, as always, meeting university colleagues, but welcomed a return to the "wide open spaces." I could not say this with free conscience next year, for this fall, at long last, a medical school is due to open at the University of British Columbia, in Vancouver.

Since the policy of this Society was changed from that of a small, restricted group to the equivalent of a National or North American Association, there has been a rapid growth of our membership. The expansion has been paralleled by an increase in the proportion of members who do not have direct affiliations with teaching or research institutions. To these men, in particular, I address my preliminary remarks.

The introduction of a new specialty in medicine to a community is seldom

* Presidential Address, under title "Travail in the Far West, or, Neurosurgery Is What You Make It," at the meeting of the Harvey Cushing Society, Colorado Springs, Colorado, April 16, 1950.

† 913 Medical-Dental Bldg., Vancouver, B. C.

the occasion for a committee of welcome. A few leaders in the local profession will say, with conviction, that "there is always room for a well trained man." They will help to set the stage for the neophyte by arranging a suitable hospital appointment and then, as a sympathetic audience, sit back to watch Act One unfold. The remainder of the doctors will appear to be singularly disinterested. They have hitherto not recognized any special need. Patients in the new category may be a rarity in their experience. Or they may always have treated, with satisfaction, the lesser conditions in the new field, and hold the conviction that what they cannot treat should go to another center, preferably far away.

One's early experience in actual practice is apt to shatter the complacency that years of sheltered post-graduate study usually provide. The first patient who appeared on my doorstep complained of blurred vision, and had some obscure form of retinal degeneration. I was almost as shocked by his name as by my failure to recognize his disease. His family name and surname were the same as a very distinguished neuropathologist. The significance of this curious coincidence I was never able to fathom. If the first surgical case had presented himself with the same name I might have heeded the obvious warning and been spared an ordeal. There was little doubt that this patient had a brain tumour but localizing evidence was not overly clear. It was a shock to find that the gallery was crowded with internes, nurses, and a generous sprinkling of staff doctors. After expertly turning down a bone flap I was unable to remove the tumour. Indeed, I could not find it. The audience would have regarded removal of that tumour as a near-miracle. Having seen one try hard and fail, they were satisfied and friendly. The decision to take up a life of logging or fishing, which had been resolved by the operator during the course of his unsuccessful endeavour, was abandoned. A career in neurosurgery was launched.

Fifteen to twenty years ago it was easier for a neurosurgeon to obtain special dispensation for equipment and operating room than it is today. There was no need for reserved beds. Most of the major neurosurgical operations were tedious, and impressive by virtue of their length. "How do you ever stand those long operations?" was the daily query of our colleagues. Only the irreverent asked "What are you doing all that time?" Nowadays, our operating time corresponds with the average major procedure in general surgery. Operation on the chest and heart may be much longer and perhaps more impressive. A bone flap for brain tumour is inconspicuous on the daily operative list of a large general hospital. Any air of grandeur which might still have lingered has been dissipated by the very serious pressure for hospital beds, which makes even our most sympathetic colleagues look with a jaundiced eye on special indulgence.

To accomplish good neurosurgery in a non-teaching general hospital, within the framework of a department of general surgery, requires a certain tenacity of purpose. Whether one works exclusively in a single, specially designed operating room, or in several different hospitals, or in six different

operating rooms in the same hospital—as I have done, one can devise and improvise a setup that is adequate. But there can be no quarter with the fundamental principles of our special craft. Firm insistence on standards of skin preparation, draping and wound closure, together with an instinct to handle tissues gently, rigid control of hemorrhage, and a keen awareness of the dangers of anoxia; this has maintained our prestige in the changing order of surgery. It has allowed us to wander with singular impunity from scalp to toe, restricted only by the convention that we are seeking nervous tissue—neurological surgeons by repute, general surgeons by design.

The ability of the well-qualified neurosurgeon to extend his surgical activities outside the confines of the skull and the spinal canal has allowed many of us to develop a satisfactory practice in a community which, so we were told, could not possibly support the services of a "brain surgeon." It has also brought us perilously close to a situation which was foreseen by Dr. Cushing many years ago. Speaking in 1913 on "Realignments in Greater Medicine,"[1] he said: " . . . the existence of the operating specialist, as contrasted with the general surgeon, is justified only if the former takes advantage of his opportunities to contribute to the knowledge of the disorders he specially treats. When progress ceases to be made, through the intensive studies which the smaller field of work permits, there is every reason why the vagrant specialty should be called back under the wing of its parent, general surgery, from whom under no circumstances should it ever be permitted to wander too far." Therein lies our quandary. How may the neurosurgeon who has no research assistants, no teaching assignments, no intensive concentration of special cases, maintain the zeal to investigate and to promote knowledge in his restricted field, failing which he deserves and will receive no special privilege?

Experience has taught me some of the negative answers to this question, and a very few answers that might qualify as positive. One has learned not to initiate bright projects, which depend chiefly for their sustenance on the activity of someone in an ancillary department. Alternatively, it takes only a few lessons to find how tiresome and ineffective is any attempt to duplicate the activities of another department in one's own person. Try making a clinical movie, all by yourself, to be persuaded of that. Unless one is an inventive fellow or a gifted craftsman with a flair for improving the mechanics of our job, the most satisfactory contribution is entirely a product of mind—the revealing consideration of a solitary case, or the fruitful synthesis of a well chosen group.

One of the outstanding exponents of this method was the late Professor Otfrid Foerster, who is remembered with affection by many of this Society. He had an unusual capacity for extracting scientific data from purely clinical observations. Sometimes it appeared to the foreign workers at his Clinic, as though he regarded all diseases of the nervous system, or operations thereon, as physiological experiments. That was, of course, only one side of his character as a doctor, but it was the most evident aspect to his pupils. The back-

ground of his special ability was an encyclopedic knowledge of the literature in neurology and neurosurgery, and particularly in neurophysiology. Professor Foerster's methods are as favorable to the circumstance of practice in an isolated general hospital as in a specialty service within a university. They are particularly adapted to the needs of a lone neurosurgeon because they depend on no one but himself.

LATER ON

Before I am tempted to overstrain your credulity I will abandon the role of venerable sage, and leave further consideration of this subject to my successors in office, who may serve as a better example. There is another aspect of a career in neurosurgery which I introduce with diffidence, although convinced that it is a matter of importance. I refer to the responsibility of the neurosurgeon to the medical profession as a whole, having in mind the problems of medical economics.

This Society was formed during a period that some dismal realists now refer to as the "Hungry Thirties." It was a time when agencies outside of medicine were beginning, in this country, to scrutinize critically the costs of medical care. Dr. Cushing was keenly aware of the far-reaching implications of the problems and became earnestly involved in the activities of a committee of national importance. The story is well told by John Fulton.[2] During 1934–35, Dr. Cushing was a member of the Medical Advisory Committee to the Committee on Economic Security. There was then being evolved in Washington the first of a series of congressional bills with which you are all probably more familiar than I. Harvey Cushing distrusted reformers outside of medicine who proposed to alter the whole pattern of medical practice. His attitude in the face of these problems was forthright, the reaction of an individualist who made up his mind about the important issues and then strove mightily to be consistent. Feeling secure to his position in the medical world, he did not hesitate to criticize what he considered to be faults in the attitude of organized medicine, at the same time that he proposed more considered thinking to the enthusiasts for extreme legislation.

It would be an affront to our colleagues in general practice and in the other specialties, to suggest that a training for neurosurgery endows the recipient with any special ability to lead medicine out of today's economic morass. But an established neurosurgeon has a position in his medical community which should make him potentially more acceptable for responsible office than many of his confreres. As a relatively lone specialist who draws patients from a wide field, he has made the close acquaintance of a very large number of doctors. He has no obvious axe to grind. Sir William Osler once said of himself that he was befriended by all medical men because he was in competition with none. The relative absence of competition and the special facilities for hospital work that are usually enjoyed by the neurosurgeon, are privileges to be repaid, if privilege is to remain.

Most of my friends in medical practice talk and act as though they would

like to forget about economics, if that were possible. They have been trained to treat the sick and want only to get on with the job. As matters concerned with the costs of medical care become more complex, they become increasingly frustrated. When their problems are brought to medical meetings it is not unusual for confusion to be further compounded. Like Mrs. Ramsbottham, whose young Albert was swallowed by the lion, they feel that "someone's got to be summonsed." They wonder whom they should trust, and vaguely hope for good leadership.

At a time when traditional ethical and social values in Western civilization are in the melting pot, it is not surprising that the claim of Medicine to remain an independent, self-governing guild should be challenged. It is perhaps more surprising to sense the unwitting faith in our organization that is implied by our demand for continuance of a large measure of self-government. Guidance of our affairs has rather suddenly become very complex. Responsible office in a general medical society can no longer be delegated as a mere token of esteem. Few of us are fitted by personality or training to withstand buffets of public criticism, the rancour of dissatisfied colleagues, or worse still, the doubts of uninformed friends. Yet this may be the lot of a leader in Medicine, today. We require men who will consistently represent the profession as a whole, men who will speak out for Medicine. They are needed, not alone in the councils of national associations, but in the smaller societies and committees of the regions and districts. They must come from all branches of medicine—not excepting neurosurgery, which has seldom shown any predilection for the ivory tower.

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INTRAMEDULLARY SPINOTHALAMIC TRACTOTOMY*

ALBERT D'ERRICO, M.D.†
Dallas, Texas

(Received for publication September 8, 1949)

IN SPITE of a definite knowledge of the location of the pain tracts in the upper levels of the nervous system, the relief of intractable pain in the upper extremities, shoulders and neck has not been uniformly satisfactory, and many procedures are still done offering incomplete relief and resulting in needless neurological deficit.

The lesions most commonly encountered are carcinoma with extension into the brachial plexus from the neck or from the breast, and superior sulcus tumors with involvement of the sympathetic chain as well as the segments of the arm. Where the pain is confined to the neck or upper chest, rhizotomy gives satisfactory relief, but a lesion involving either the segments of the neck or those of the upper chest will usually at some time involve the segments of the upper extremity. The disability of the upper extremity resulting from a rhizotomy is so great that a more selective operation, even at greater risk, would be desirable for the patient, and a section of the spinothalamic tract sufficiently high to include the distribution of the pain is obviously necessary. High cervical chordotomy has been employed, but with many unsatisfactory results. The level resulting is often not sufficiently high to include the area of pain and in addition so many of these patients have pulmonary involvement that damage to the phrenic segments may be particularly dangerous. This necessitates a still higher section of the spinothalamic tract.

The procedure of Schwartz and O'Leary[1] sectioning the spinothalamic tract in the medulla at the level of the lower border of the olive appears ideal, but the paucity of reports on this procedure indicates that it has not had wide acceptance. This lack of acceptance has apparently been for three reasons, (1) the incomplete sensory loss of the shoulder and neck, (2) the technical difficulties of the procedure itself, and (3) the reported postoperative ataxia in ambulatory patients.

Twelve cases of intramedullary spinothalamic tractotomy for intractable pain of the neck, shoulder and upper extremities are reported here, and our experience with these cases has proven these difficulties to be largely invalid, so that the procedure is now considered a most satisfactory one. In the early cases, incomplete sensory loss in the shoulder and neck was the rule, but with further mapping of the tract it has been possible to obtain higher and more satisfactory levels of analgesia. According to Schwartz and O'Leary, and also Walker,[2] there is a topographical arrangement of the pain

* Presented before the Harvey Cushing Society, San Francisco, California, August 19, 1948.

† 510 Medical Arts Building, Dallas 1, Texas.

and temperature fibers which lie between the dorsal border of the inferior olive and the 10th nerve filaments, the sacral segments lying most superficially and the cervical deepest (Fig. 1). On our first test case, a narrow cut skirting the olive was carried in stages to a depth of 6.5 mm. A level of analgesia up to the nipple line was produced at 5 mm., but this did not rise with the additional cut of 1.5 mm. A Horner's syndrome was produced as the cut was carried to its deepest extent. The incision was then carried to the level of the 10th nerve filaments, and a small rise of analgesia to include the shoulder was obtained together with an hypalgesia of the neck. The incision was then carried 1 mm. more dorsally still, and immediate analgesia was obtained to a level slightly above the border of the mandible. As the incision was carried posterior to the level of the vagus filaments, the patient complained of sharp pain on the ipsilateral side of the face. In succeeding cases, it was found necessary to extend the cut posteriorly to a level where pain on the ipsilateral side of the face was felt in order to obtain the highest analgesia possible. The curve of the knife blade does not permit a selective deep dorsal cut without carrying the superficial portion of the incision into the trigeminal tract. It is possible, therefore, that the elusive pain fibers to the neck lie deep and dorsal. The incisional depth which proved the most satisfactory was 5 mm. In practically every instance, analgesia was obtained to a level above the mandible and to the vertex posteriorly at the time of operation, but in some cases this level receded slightly in several weeks with a moderate hypesthesia remaining in the upper portion of the neck (Figs. 2 and 3).

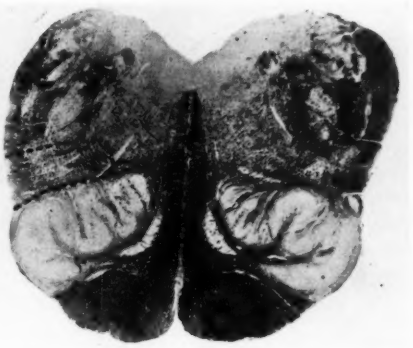

FIG. 1. Section through inferior olive showing extent of initial cut; higher sensory level as necessary obtained by more dorsal cutting maintaining same depth.

These findings appeared to be borne out in the reports of White,[3] and Schwartz and O'Leary. The fact that White obtained about as high a level with a 4 mm. cut as Schwartz did with a 6 mm. cut appears significant. In the case of White, analgesia was obtained up to the shoulder, but only an hypalgesia resulted over the shoulder and neck. This was obtained with a cut between the levels of the vagus nerve and the olive at a depth of 4 mm. In the first case of Schwartz and O'Leary an incision 6 mm. deep between the levels of the vagus filaments and the olive produced an analgesia up to the clavicle. In their second case, an incisional depth of 5 mm. produced analgesia up to the 1st dorsal segment. There was one finding in White's operative report which appears significant, fitting in with our findings, and this was the fact that he produced pain on the ipsilateral side of the face with

his cut, whereas this was not mentioned in the operative reports of Schwartz.

The technical difficulties of operation are sometimes great, due chiefly to the application of branches of the posterior inferior cerebellar artery to the surface of the medulla at the site of the incision. A great many of the technical difficulties can be overcome by proper positioning of the patient and the use of an altered, curved #12 Bard Parker blade used with the long #7 handle.

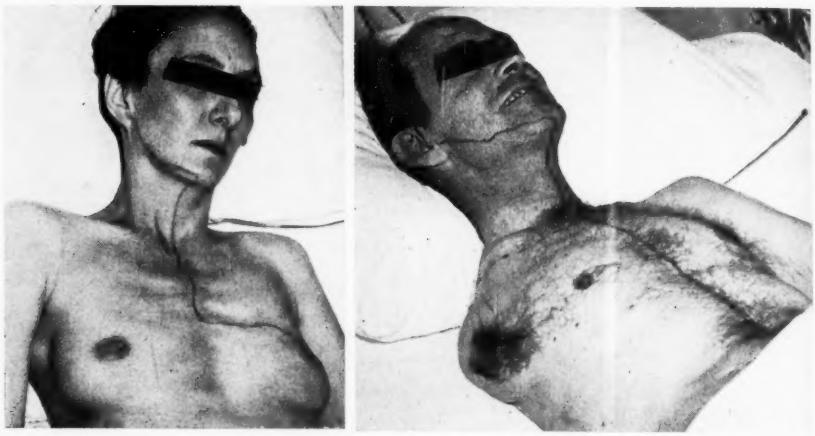

FIG. 2 (*left*). Typical high level of analgesia by intramedullary spinothalamic tractotomy on patient's right contrasted with level of analgesia on the left obtained by later chordotomy at T1 for subsequent spinal metastasis.

FIG. 3 (*right*). Characteristic sensory pattern with incomplete dorsal cutting showing hypesthesia between dashed line below the clavicle and solid line above the mandible, and analgesia below dashed line.

The proximal portion of the cutting edge is ground away leaving exactly 5 mm. distally. A black line is inked across the blade at a depth of 5 mm. using laundry ink which sets into the metal and does not come off with soaking (Fig. 4). The curve of the blade provides good access for the incision and the black line a good control of the depth. With the patient in the sitting position, much better exposure and vision can be obtained with a far more desirable working position. In some cases, these advantages appeared sufficient to enable the operator to make the incision where otherwise he would not. A unilateral flap with removal of a portion of the arch of the foramen magnum is used, since a wider approach to the region can be obtained. In almost every case the vessels could be teased away from the site of the incision just caudal to the 10th nerve, although in 2 cases the incision

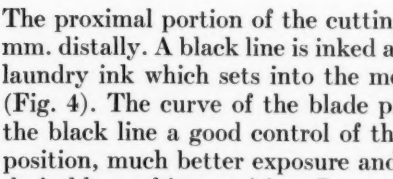

FIG. 4. Photograph showing knife blade used with edge ground off up to laundry ink marking at 5 mm. from tip.

had to be made obtaining access to the tract between the filaments of the 10th nerve. Marked hoarseness at the time of operation followed, but this improved greatly by the time of closure and had disappeared almost completely within a period of 2 weeks. Some hoarseness followed in other cases, but this was only very temporary. The operation obviously must be done under local anesthesia.

RESULTS

The condition of most of the patients was poor, and only 3 were able to walk before operation. Following surgery, these showed ataxia as described by White, but they were able to walk unassisted after a period of 2 months from operation. Two stated that during the 3rd and 4th postoperative weeks they could get about only by crawling, but within a month both could walk assisted, and within 2 months unassisted. By the end of 3 months, scarcely any incoordination in walking could be observed. Schwartz stated that cerebellar disturbances were not present in his second patient, but he also noted that a pathological fracture of the left hip prevented testing coordination in walking. In all of our cases, there was little or no cerebellar disturbance demonstrable while the patient was lying in bed, but it was marked on first attempting to get up. This ataxia is difficult to explain since the incision is far from the restiform body. Dividing the ventral spinocerebellar tract cannot be considered a cause since this tract is necessarily severed with a chordotomy where no such ataxia follows. It may be due to trauma to the olivary nucleus.

Of the 12 cases, 6 were carcinomas of the upper lobe of the lung. There were 3 carcinomas in the neck and 1 carcinoma of the breast with extension into the brachial plexus. There was 1 luetic neuritis and 1 amputation neuralgia of the right arm. Complete relief was obtained by every patient with lung tumor even though some of these had already been addicted to narcotics. The patient with carcinoma of the breast obtained complete analgesia over the area of her pain, but when narcotics were withdrawn she complained of pain. She again became free of pain with moderate doses of narcotics given regularly, whereas before operation large doses had given no relief. The case of luetic neuritis presented a similar problem due to narcotic addiction. The patient with amputation neuralgia obtained complete relief of his excruciating pain in the axilla and complained only of some tenderness in the upper neck. In the first case of carcinoma of the neck involving the brachial plexus complete relief of arm and shoulder pain was obtained. The second patient with carcinoma of the neck obtained relief of neck pain but pain developed later in the 3rd division of the 5th nerve on the same side. The third patient with carcinoma of the neck expired immediately following operation, presumably from a coronary thrombosis. This was the only mortality of the series. All of the other patients tolerated the operation extremely well and showed no alarming ill effects.

SUMMARY

Twelve cases of intramedullary spinothalamic tractotomy are reported with satisfactory relief of pain and without lasting disability in 11 surviving patients.

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A STUDY OF 182 PATIENTS WITH VERIFIED ASTROCYTOMA, ASTROBLASTOMA AND OLIGODENDROGLIOMA OF THE BRAIN

LOYAL DAVIS, M.D., JOHN MARTIN, M.D., FRANK PADBERG, M.D., AND ROBERT K. ANDERSON, M.D.

*Department of Surgery, Northwestern University Medical School, Chicago, Illinois**

(Received for publication November 7, 1949)

Statistics are dreary matters but it is periodically incumbent upon us to assemble our cases not only for our own instruction lest we bury in obscurity our mistaken and bad results, but also to acquaint others with the standing of operative measures.—HARVEY CUSHING

FROM July 1, 1924 to January 1, 1949, 972 intracranial tumors have been verified microscopically. Of this number, 523 (53.8 per cent) were classified as gliomas; of these, 160 (16.6 per cent) were astrocytomas, 24 (2.5 per cent) were oligodendrogliomas and 5 (0.51 per cent) were astroblastomas. In each case the diagnosis has been verified and the classification made by microscopic sections according to established histopathologic criteria. Comparable studies of the glioblastomas and the medulloblastomas have previously been published.[7,8]

ASTROCYTOMAS

Employing the classification outlined by Bailey and Cushing,[3] we have subdivided the astrocytomas into *fibrillary* and *protoplasmic* types according to the predominating cellular structure (Figs. 1 and 2). In a few instances tumors with a structure intermediary between these types have been designated as *mixed* (Fig. 3). Some of the tumors were solid, others were cystic, and still others were situated on one wall of a large cystic cavity. There were 25 astrocytomas located in the cerebellum; the remaining 135 were in the cerebrum. Of the total 160 patients, 153 have been followed and form the basis of this study (Table 1).

Cerebral Fibrillary Astrocytomas

The survival time following surgical treatment of the cerebral fibrillary astrocytomas, 114 in number, cannot be correlated with the age, sex or duration of symptoms. The longest survival record of 243 months is held by a female patient who was 27 years of age at the time of her operation and who had had symptoms for 3 weeks previously. Thirteen patients (6 males and 7 females) have survived 61 months after operation and their average age was 33 years at the time of operation. Six of the 13 patients had experienced preoperative symptoms for 2 months or less, 12 for less than 4 months. Of the patients who lived less than 60 months after operation 101 had a

* Aided by the Grunow Surgical Fund.

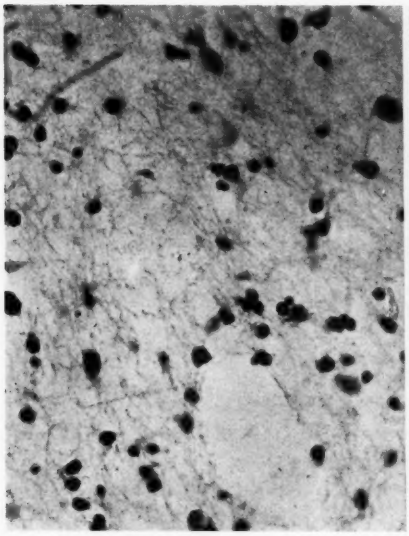

FIG. 1. Fibrillary astrocytoma. (Hematoxylin-eosin, ×220.)

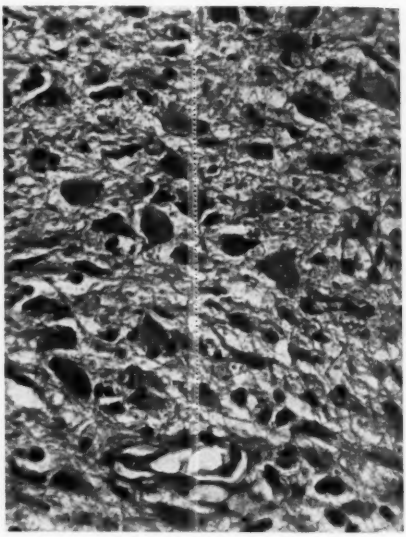

FIG. 2. Protoplasmic astrocytoma. (Hematoxylin-eosin, ×220.)

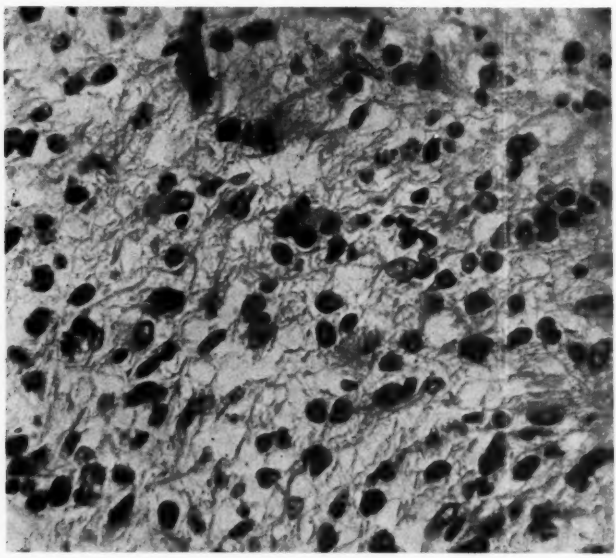

FIG. 3. Mixed astrocytoma. (Hematoxylin-eosin, ×220.)

range in duration of the preoperative symptoms between 3 weeks and 21 years. Four of the 101 patients had recognizable symptoms for only 6 months or less before operation; 15 survived 1 month or less and 38 for 6 months or less after operation.

Sixty-two (54.3 per cent) of these patients had convulsive seizures prior to operation. There was a low incidence of seizures in the patients who survived operation for short periods and conversely a higher incidence of seizures with a longer survival period. However, the 3 patients with the

TABLE 1

SURVIVAL TIME IN YEARS

	No of Patients	0-½	½-1	1-2	2-3	3-4	4-5	5-10	10-15	15-20	20-25	Social and economic independence
Astrocytomas Cerebral fibrillary	114	38	17	18	13	11	4	7	3	2	1	37
Protoplasmic	9	3	0	2	2	1		1				2
Mixed	5	2	0	2	1							2
Astrocytomas Cerebellar fibrillary	22	5	2	2	2		1	2	4	4		8
Protoplasmic	1	1										
Mixed	2							1		1		1
Astroblastomas	5	4	1									
Oligodendrogliomas	24	7	3	1	5	4	3				1	8

Table 1

longest recorded survival gave no history of convulsive seizures at any time during the course of their illness.

Calcification of the tumor might well be taken as evidence of nature's attempt to reduce the rapidity of the growth of the tumor. In this group, 14 patients (12.3 per cent) had calcification of the tumor recognizable upon the x-ray films, but the character of the calcification or its distribution was not significantly diagnostic of the histological nature of the growth. Symptoms had been present in these 14 patients from 1 month to 10 years prior to operation, and the survival periods after operation varied from 3 days to 66 months. Sixty of the fibrillary astrocytomas were located in the right and 46 in the left cerebral hemisphere; 7 tumors involved the corpus callosum and 1 was found in the third ventricle and midbrain. The parietal lobe was most frequently involved; the frontal lobe was often involved with the temporal lobe, and the occipital lobe contained tumor tissue in only 5 cases. Fifty-four of the tumors were solid; 47 were multicystic and only 13 contained a large cyst or multiple large cysts with smooth-walled cavities and a mural nodule. The patient with the longest recorded postoperative

survival period, 243 months, had a solid fibrillary tumor removed, but the average survival time of the patients with similar tumors was 26.4 months as compared with 29.5 months in cases of multicystic tumors, and 42 months in those instances in which large cystic masses were found. One patient died $14\frac{1}{2}$ years after removal of a large cystic astrocytoma; another is attending college 9 years and 10 months following operation, and 3 others are at work and asymptomatic 26, 30 and 34 months after removal of their tumors. In our series, the longest recorded survival time of a patient with a glioblastoma was 41 months and of a patient with a medulloblastoma, 36 months.[7,8]

There was a great variation in microscopic structure of these tumors, irrespective of their gross appearance. Some were composed of adult astrocytes with round, or oval, darkly stained nuclei arranged without pattern and surrounded by abundant microglia, and without any significant alteration of the blood vessels (Fig. 1). Others contained fibrillary astrocytes, numerous protoplasmic astrocytes, and some oligodendroglia. The protoplasmic astrocytes (Fig. 2) varied in size and showed occasional mitotic figures and deeply stained nuclei. Marked cellularity, increased vascularity and microcystic changes were often present regardless of whether the tumor at operation was firm and solid, stringy and tough, or soft and mushy. We have been completely unable to correlate these histopathological changes with the survival time of the patient, or to state categorically that a cystic astrocytoma is *prima facie* evidence of a more favorable prognosis following operation, although certainly this is the clinical impression one gains. However, there is definite evidence that patients with large astrocytomatous cysts have a longer survival period following a complete surgical excision of the cystic masses. It was this group of tumors which gave encouragement to general surgeons in the past when they "tapped a brain cyst," and there is in our series the record of a patient with a cystic cerebellar tumor who had been so treated 20 years before a recurrence, at which time the nodule in the cyst was removed *en bloc*.

We have been unable to define histologically separate groups and subgroups among the fibrillary astrocytomas as have Alpers and Rowe.[1] We are in agreement with Scherer's study[12] in which he emphasizes the importance of examining many sections from representative areas of the tumor, particularly since we have 3 instances of microscopic evidence of what originally appeared to be a fibrillary astrocytoma changing to a rapidly growing glioblastoma. Cairns[5] has called attention to the considerable variation in the rate of growth of the individual astrocytomas, and we raise the question as to whether they would have been diagnosed astrocytomas if the entire tumor mass had been available for microscopic study in the beginning.

Eight of the 114 patients with fibrillary astrocytomas of the cerebrum were treated with a radium bomb. In most instances the factors were as follows: Distance 10 cm.; 2 portals, each 8×10 cm.; dosage 2,000 mg. hours of exposure through a 2 mm. equivalent brass filter. Seven received from 13,000 to 160,000 mg. hours of irradiation and 1 patient received 3 series of

treatment totalling 350,000 mg. hours. One of the patients who received 13,000 mg. hours survived for 40 months and the one who received 350,000 mg. hours survived 43 months. Microscopic sections of the tumors of these 2 patients made following irradiation by this method showed many areas of necrosis and cystic degeneration with some fibrotic repair and numerous thickened, tortuous blood vessels. Mitotic figures were not seen. The longest survival period following this type of radiation therapy was 123 months and

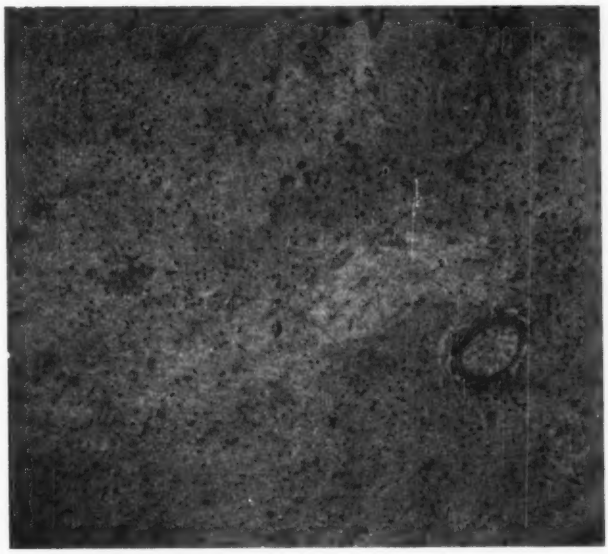

FIG. 4. Fibrillary astrocytoma after x-ray therapy; operative specimen. (Hematoxylin-eosin, ×50.)

the average was 36.5 months, with no correlation between the number of mg. hours of radiation and survival time.

Seven of these 114 patients received x-ray therapy postoperatively varying from 1,600 *r* in air to 14,438 *r* in air. In most instances the factors were as follows: 200 kV.; 1 mm. copper and 1 mm. aluminum filters; distance 50 cm.; 3 to 5 ports, depending on the location of the tumor. The average survival time for these patients was 39 months, varying from 6 to 67 months. This is 9 months longer than the average survival time for all of the patients in the series, which was 29.9 months, regardless of the methods of treatment.

Microscopic sections of these 7 tumors made before and after x-ray therapy were studied. Definite changes were observed, consisting of ring-like, bizarre formations of cells and clumps of broken-down hyperchromatic nuclei, hyalinization of both stroma and blood vessels, and a preponderance of thickened, occasionally thrombosed, tortuous blood vessels with a marked proliferation of the intima which almost obliterated the lumen. There were

many areas of necrosis, some of which showed fibrotic repair. In some areas frank liquid necrosis was present (Fig. 4). These changes were inconstant and showed considerable individual variation. There was no correlation between the number of *r* in air administered, the post-radiation microscopic picture, and the survival period.

There were 32 other patients of this series treated with x-ray irradiation postoperatively, but microscopic sections of the tumors after radiation were not available. These patients received an average of 5,236 *r* in air. The smallest amount given was 660 *r* and the greatest, 16,750 *r*. The patient with the longest survival period (243 months) received 1,200 *r* in air. The average survival period in this particular group of fibrillary astrocytomas was 43 months, which is 13.1 months longer than the average for all the tumors in this group regardless of methods of treatment.

Three other patients were treated with radium bomb therapy followed by x-ray irradiation. Their average survival period was 29 months. One of these, who survived 51 months, received 7,672 *r* in air by x-ray radiation and irradiation from radon seeds which were implanted into the site of the tumor removal. Autopsy specimens of the tumor showed many distorted blood vessels with thickened walls, areas of necrosis with fairly good fibrotic repair and a great variety in size and shape of the nuclei, which contained no mitotic figures.

Bailey, Sosman and Van Dessel[4] stated: "These gliomas vary so much in histological structure, and the degenerative changes which occur spontaneously are so frequent that it has been practically impossible to recognize any constant alteration in the microscopical structure of any type of glioma which we were sure was due to roentgen treatments." Frazier and Alpers[9] catalogued the effects of x-ray radiation on astrocytomas as a definite transition of less mature elements to mature astrocytes, an increase in the number of undifferentiated multinucleated giant cells, an increase in extent of areas of necrosis and degeneration of the stroma, thickening of the intima of blood vessels, and small intimal hemorrhages and hyaline changes in all coats of the vessels. We do not believe we can estimate the number or extent of pathological changes possible, nor the conditions of transition from a phase of rapid to slow growth in the natural development of these tumors. The results of irradiation therapy are therefore equivocal at best, and no hard and fast rule can be laid down regarding the indications or ultimate results of this therapy.

We have always attempted to remove gliomas as completely as possible, consistent with preservation of the motor centers and speech area of the dominant hemisphere. Our craniotomy exposures have been large and resection of the tumor has been followed by partial or complete removal of the bone flap. There were 196 surgical operations performed upon the 114 patients with fibrillary astrocytomas, 29 of whom succumbed in the hospital at varying periods after operation. Regardless of the length of the interval, they are computed as postoperative deaths. The results in some of these cases

were obtained when silver clips, the suction apparatus, and older methods of anesthesia were the only surgical aids, but they must be compared to those of the last half of the years in our experience during which gelfoam and other hemostatic agents, the high frequency scalpel, blood transfusion facilities, newer anesthetic agents, and the antibiotics were at our disposal. In this series we consider that 37 patients have been able to pursue socially useful lives after surgical treatment. A larger number of patients were discharged from the hospital symptomatically and physically improved, but they were unable to return to their previous occupations.

Cerebral Protoplasmic Astrocytomas

Our series includes 9 cerebral protoplasmic astrocytomas. The longest postoperative survival period was 44 months, the average being 19 months. Two patients died postoperatively and 2 were considered to have led useful lives after operation. One patient gave a history of convulsive seizures for 7 years before operation, but all the other patients had had symptoms for about 6 months. One had as her only complaint a single convulsive seizure, but there was other evidence of an organic lesion. She refused operation and without any treatment whatever survived for 6 years and 4 months from the onset of her symptoms. Eventually there developed mental and speech changes, a right hemiplegia and symptoms of increased intracranial pressure. At autopsy the tumor was found to have invaded the entire left cerebral hemisphere.

Six of these tumors were multicystic and extensive, and they infiltrated large areas of the brain. A second surgical procedure was performed upon only 1 patient. Originally this tumor was firm, presented on the cortex of the right frontal lobe and was surrounded by softened brain. At the second operation, 27 months later, the tumor had extended into the left frontal lobe. An extensive resection was performed and a large cyst was aspirated in the right frontal lobe.

Three patients in this group received x-ray irradiation, which varied in amount from 3,200 to 5,924 *r* in air. The patient who received 3,200 *r* in air was also treated by implantation of radon seeds into the site of the tumor removal.

Bailey, Sosman and Van Dessel[4] have described these tumors as generally slow growing with occasional mitotic figures and numerous amitoses. They stated that differentiation was possible in these tumors.

Mixed Cerebral Astrocytomas

Five of the 128 cerebral astrocytomas were classified as *mixed* because histologically they were neither predominantly fibrillary or protoplasmic (Fig. 3). There were 8 surgical procedures upon these 5 patients without a postoperative fatality. The longest survival period in this group was 30 months. One patient is still alive 25 months after his first operation. Preoperative symptoms varied from 1 month to 6 years and 2 patients com-

plained of convulsive seizures only. A wide surgical resection was performed in all of these cases. Three of the tumors were solid, and 2 were multicystic. Two of the patients were able to lead useful lives; one managed his farm and worked vigorously for 12 months, and the other worked productively for 27 months.

The patient who survived 30 months had a wide, but incomplete, resection of a left parietal lobe tumor. He received 5,544 *r* in air of x-ray irradiation postoperatively and was able to work and cared for his family for 29 months. At autopsy, an extensive tumor was found which involved the corpus callosum, both frontal lobes, and the pons. Microscopic examination of several areas of the tumor did not reveal any demonstrable microscopic effects of irradiation.

Cerebellar Astrocytomas

There were 25 astrocytomas located in the cerebellum chiefly of children and young adults whose average age upon admission to the hospital was 16 years. Eleven patients were less than 15 years of age and 3 were over 40.

The progress of the growth of these tumors was found to be usually slow. There was a fairly long history prior to admission with an average duration of symptoms of 14 months. In children, a long history of recurrent morning headaches and vomiting often was uninvestigated because in between such episodes they were apparently well and the complaints were often considered to have arisen from faulty function of the gastro-intestinal tract. In adults, instability in gait and awkwardness in the use of the upper extremities were common early complaints.

The cerebellar astrocytomas produce essentially the signs of midline lesions, and they exist predominantly as purplish nodules of varying size which project from the wall of a cystic cavity. The fluid in the cavity varies in color from light yellow to brown and has a high protein content. The fluid probably is the result of transudation of serum from the vascular surface of the tumor nodule. Many of these tumors involve the vermis of the cerebellum primarily. There were 5 such cases in our series. In the remaining instances, where large cysts were found in either cerebellar hemisphere, with but 1 exception the tumor nodule was located medially and the cyst had expanded laterally into the hemisphere.

Cytologically, all except 3 of the 25 tumors were of the fibrillary type. Two demonstrated about equal proportions of fibrillary and protoplasmic cells and were classified as mixed astrocytomas; 1 was predominantly protoplasmic.

Fourteen patients received postoperative x-ray irradiation consisting of at least 4,000 *r* in air; 5 received over 10,000 *r* in air over a period of years. The remaining 11 received no irradiation of any type. In the treated cases there are 7 patients who are still alive after operation from 6 months to 18 years ago. In the untreated group there are 7 alive who were operated upon from 2 to 16 years ago. There were recurrences in 4 patients who received

x-ray irradiation but 3 of these patients were known to have had resections which were, of necessity, incomplete. It is our opinion that x-ray irradiation has little, if any, effect on the cerebellar astrocytomas and this is in accordance with the observations of others.[4,6,11]

The adverse effect of pregnancy upon tumors is emphasized in 2 of these patients. One, a 22-year-old married woman, had an acute onset of the general symptoms of a space-occupying lesion and soon after, during her last trimester of pregnancy, specific cerebellar signs developed. Twenty days after delivery of a normal infant, she was operated upon and a large cyst was found in the vermis which extended into the right cerebellar hemisphere. In the depths of the cyst toward the midline, a mural nodule was found and removed. The second was a 17-year-old girl from whom a cerebellar tumor had apparently been completely removed from the vermis and right cerebellar hemisphere at the age of 8 years. She had made an excellent recovery and had no evidence of cerebellar dysfunction. At the age of 17 she became pregnant. The pregnancy was terminated by abortion, but headaches that had developed during this period persisted and became more severe. Again nausea, vomiting and loss of coordination occurred. The area of suboccipital decompression, which had remained flat during the 9 years following operation, became elevated and tense. A second operation revealed a large cyst and mural nodule, involving the vermis and both cerebellar hemispheres. It would appear that the pregnancy precipitated, or at least stimulated, the advance of the growth of relatively latent tumor cells. It is our custom now to advise young married women who have had intracranial tumors removed not to become pregnant, and in 2 instances surgical sterilization has been performed.

Ten of these patients, some of whom were operated upon 17 years ago, have returned to their former occupations and social positions and continue to be well at this time. Five made excellent recoveries and returned to their former positions but then recurrences developed 8 to 12 years after operation. There were 4 patients with far advanced symptoms of cerebellar dysfunction at the time of operation who improved following surgery, but who were not able to make a satisfactory rehabilitation with respect to their former social and economic positions. They are, however, still alive from 2 to 10.5 years postoperatively.

There were 7 deaths in the hospital among this group of 25 patients. One 3-year-old child expired while under observation without having been operated upon. In 23 primary cerebellar operations, there were 4 deaths with an operative mortality of 17.4 per cent, but the increased risk involved in secondary operations upon the cerebellum in this group of tumors is indicated by 2 deaths in 8 operations.

ASTROBLASTOMAS

We have reserved for this classification those tumors in which astroblasts are the predominating cell type, although many astrocytomas and

glioblastomas contain astroblasts in varying numbers. Five tumors in our series satisfied our microscopic requirements, and all were found in the cerebral hemispheres. They were diffusely infiltrating tumors and only 1 showed a tendency toward limitation of its gross boundaries. This was a cystic tumor, but the others were invasive and contained necrotic and hemorrhagic areas.

Microscopically, the stroma is very cellular with reticulated areas between the clumps of cells. The astroblast has a triangular cell body with an eccentrically placed nucleus, round or oval in shape. One or two large coarse processes extend outward from the cell body and often are seen to terminate as perivascular "sucker feet." The cells tend to be arranged in closely packed palisades along the margins of small blood vessels. There are a few mitoses

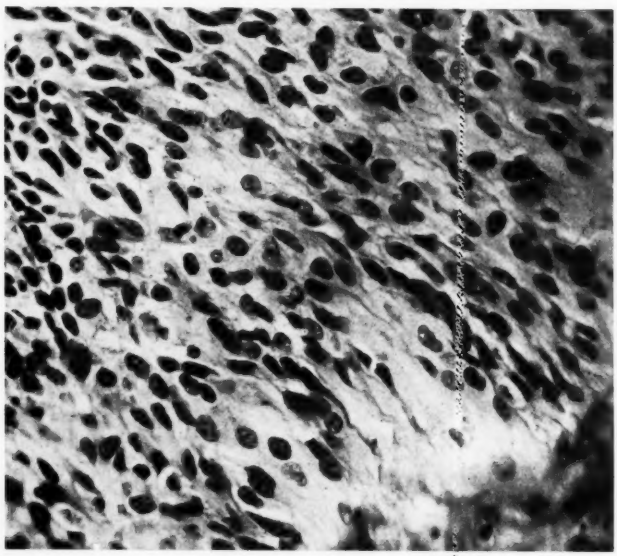

FIG. 5. Astroblastoma. (Hematoxylin-eosin, ×300.)

and the cells often contain multiple hyperchromatic nuclei. Blood vessels with moderately thickened walls are present in most of the tumor sections (Fig. 5). Bailey and Bucy[2] ascribe hypertrophy of the walls of the blood vessels in these tumors to an increase in the number of pia-arachnoidal cells around the vessels and to occasional proliferation of the intima by endothelial cells. They also state that these tumors are not a discrete type in all respects but frequently show gradual transitions to the glioblastoma multiforme, the ependymoblastoma and the astrocytoma.

In 4 of our cases the tumor invaded the cortex and a wide resection was performed. One patient died of lobar pneumonia postoperatively and examination of the formalin-fixed brain revealed no remaining tumor. Another

survived 6 months after removal of 150 gm. of tumor from the left frontal lobe. The tumor recurred and grew through the anterior limb of the scalp incision. Two patients died within the first 24 postoperative hours. Another died on the day of admission before surgery could be performed. The autopsy demonstrated a large tumor which had invaded the cortex of the right parietal lobe. Her tumor was moderately discrete and contained several large cystic masses.

OLIGODENDROGLIOMAS

There were 24 oligodendrogliomas in our series of 523 verified gliomas. Preoperative symptoms existed from 7 weeks to 17 years, and in 14 cases roentgenograms of the skull demonstrated an intracerebral calcification. In 14, the first symptom was a convulsive seizure which occurred for the first time in adult life. In our opinion, this is an important sign pointing to the presence of a space-occupying lesion. List[10] reported that in a series of gliomas the highest incidence of convulsive seizures was found to be associated with the oligodendrogliomas. Of his 33 patients 25 (75.8 per cent) complained of seizures. He concluded, therefore, that patients with calcified tumors and those with diffusely infiltrating tumors were subject to more frequent seizures. Our series substantiates this finding. Schwartz[13] after studying preoperative roentgenograms of his patients with oligodendrogliomas stated that 80 per cent showed some calcification. In our series 14, or 58 per cent, showed calcification.

Grossly, the tumors were invasive and a line of demarcation at the periphery was difficult to establish. In 14 cases the cortex was involved by the tumor and 6 tumors contained large cysts. Autopsy specimens showed that the hemisphere involved by the tumor was usually enlarged due to an increase in the bulk of the white matter. The basal ganglia and internal capsule frequently were involved by the tumor, and areas of necrosis and cystic degeneration both large and small were scattered through the tumor. The periphery of the tumor was usually firm and pale in contrast to the normal brain. Microscopically, the cells were small, contained a small, round, compact, deeply staining nucleus and a moderate amount of pale cytoplasm. The cells tended to occur in masses or clumps, to the complete exclusion of other cells in the entire field and to arrange themselves in a characteristic architectural pattern (Fig. 6). Scattered masses of astrocytes were often seen. Mitosis was very rare and amitosis was the rule. Small flecks of calcium, large areas of necrosis, and all stages of nuclear disintegration were found scattered through many of the sections. Some blood vessels showed hyalinization and endothelial thickening, and some were plugged with tumor cells. There was a great variety in the vascular pattern in the same tumor from field to field.

The survival period in this group varied from 1 to 288 months postoperatively; the average survival was 49.7 months and the average duration of preoperative symptoms was 72 months. Thirty-nine operations were performed upon the 24 patients, 7 of whom died in the hospital at varying intervals postoperatively.

From our own experience, we cannot state that these tumors tend to recur rapidly after operation. We have been unable to make a diagnosis of oligodendroglioblastoma from any of the sections in these 24 cases. Two patients did have a rapid recurrence of the tumor but these same patients gave a history of short duration of symptoms prior to operation. One patient had a progressive left hemiparesis for 6 months and left Jacksonian seizures for 4 months. At operation an oligodendroglioma was removed from the right parietal area. After operation he regained complete control of his left arm and leg, was free of convulsive seizures, and went back to work. One year later he had a seizure which was followed by weakness of his left-sided extremities. At the second operation, a large amount of recurrent tumor tissue was removed and the bone flap was sacrificed. This tumor had many young cell forms but the predominating cell type was a mature cell, rich in chromatin but without mitotic figures. Many tiny flecks of calcium were present in the stroma. The patient succumbed 9 months after his second operation. We have been unable to find any changes in the character of the microscopic appearance of these tumors following x-ray irradiation.

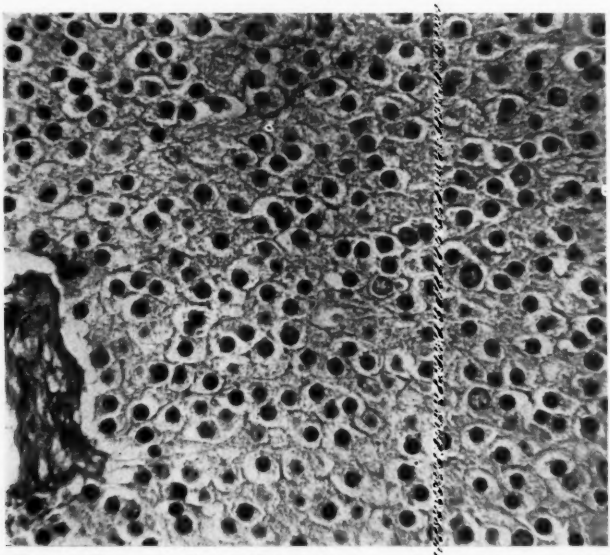

FIG. 6. Oligodendroglioma. (Phosphotungstic acid-hematoxylin, ×220.)

Eight patients in this group led a useful life during their survival period, and the patient with the longest survival, 288 months, is still alive and economically and socially independent.

It is our opinion that the histological cell type of these gliomatous tumors, the astrocytomas included, are laid down early in the life history of the tumor, and that the cellular type of the tumor remains the same.

Apparent changes in the cellular type seem to occur as a result of not obtaining a wide sampling from the tumor at the time of the initial classification. This leads to a mistaken diagnosis.

SUMMARY

We have reviewed and classified 153 intracranial astrocytomas, subdividing them as protoplasmic, fibrillary or mixed on the basis of their predominant cellular structure. Of the cerebral fibrillary astrocytomas, 54 were grossly solid, 47 were multicystic, and 13 contained large cystic masses. The patients with the tumors that were composed of cystic masses had a longer survival time than did those with the other types. We are therefore of the opinion that grossly solid astrocytomas contain more actively proliferating neoplastic cells, and that with such tumors the survival period is statistically shorter.

It is our belief that x-ray irradiation has very little effect on this general group of tumors, but definite histopathological changes followed postoperative x-ray irradiation in certain of the fibrillary astrocytomas. We do not believe that x-ray irradiation promoted growth or de-differentiation in these tumors from one type to another.

We have seen so little tendency to de-differentiation, especially in the fibrillary astrocytomas and the oligodendrogliomas, that we feel the cell type of the tumor is determined early. It is possible that some of the operative specimens of these tumors from patients with short survival periods, may have been from glioblastomas rather than astrocytomas. This could have been learned on wider sampling.

Thirty-seven of our 114 patients who had cerebral fibrillary astrocytomas, 2 of 9 patients with cerebral protoplasmic astrocytomas, 2 of 5 patients with mixed cerebral astrocytomas, and 9 of 25 patients with cerebellar astrocytomas postoperatively led a useful life and acquired social and economic independence. This, therefore, is evidence of the value of a determined surgical treatment of these tumors, malignant to a degree, and admittedly prone to recur after any form of treatment.

We have classified 5 intracranial tumors as astroblastomas, all of which were located in the cerebrum of adult patients. Four of the tumors had invaded the cortex at the time of the initial operative exposure.

It is our opinion that pregnancy stimulates the growth of intracranial gliomas, and we present evidence to substantiate this opinion in 2 of 10 female patients with cerebellar astrocytomas.

The first symptom in 14 of the patients with oligodendrogliomas in our series was a convulsive seizure which occurred for the first time in adult life. Intracerebral calcification was demonstrated roentgenographically in 14 of the 24 cases. From a review of the microscopic characteristics of these 24 oligodendrogliomas, we have not been able to make a separate classifica-

of oligodendroglioblastoma.

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INTERHEMISPHERAL SUBDURAL SUPPURATION

CARL F. LIST, M.D.*
Grand Rapids, Michigan

(Received for publication November 7, 1949)

EVEN though sporadic case reports of subdural suppuration have appeared in the literature for more than a century, accurate knowledge of its clinical symptomatology and pathogenesis is of relatively recent date and must be credited to the work of Courville,[1] Kubik and Adams,[5] and Ray and Parsons.[6] The management of this disease, which long remained in a twilight zone between the fields of otorhinolaryngology and neurosurgery, met with almost universal failure until, after the advent of antibiotics, numerous cured cases were reported (Gurdjian and Webster,[2] Schiller, Cairns and Russell,[7] Keith and McKenzie[4]). Yet in spite of all advances made, much remains to be learned about diagnosis and treatment of this puzzling condition.

In a pathologic anatomic study of 44 cases, Courville[1] found that, as a rule, the subdural exudate covered the convexity of the cerebral hemisphere. The purulent process was only rarely located in the interhemispheral fissure. More recent observations of Schiller, Cairns and Russell,[7] and of Keith and McKenzie[4] show, however, that interhemispheral site of subdural suppuration is far more common than previously realized. Failure to recognize this special situation probably explains why the mortality rate of subdural empyema has remained unduly high. It was, therefore, considered of interest to report in detail a typical example of interhemispheral subdural suppuration and to discuss pertinent problems of diagnosis and management of such cases.

CASE REPORT

Summary. Right-sided pansinusitis of 2 weeks' duration, followed by acute subdural empyema of right interhemispheral fissure; combined systemic penicillin and streptomycin therapy and drainage of empyema through three parasagittal trephine holes, with subsequent penicillin instillation. Three months later, signs of recurrent right occipital abscess; osteoplastic craniotomy with excision of chronic parieto-occipital interhemispheral subdural abscess. Second recurrence 1 month later; temporo-occipital osteoplastic craniotomy with excision of cortical occipital lobe abscess and extensive subdural granuloma of the epitentorial space. Cure verified by ventriculography; left homonymous hemianopsia only neurologic residual.

Blodgett Hospital No. 85369. C.P., 15-year-old male, referred by Dr. S. A. Fiegel, Sturgis, Michigan. First admission: March 23, 1947. Final discharge: March 14, 1948.

First Admission

History. For 2 weeks the patient had had acute right frontal sinusitis. Five days before admission he complained of severe frontal headaches associated with nausea,

* 26 Sheldon Avenue, Grand Rapids 2, Michigan.

vomiting and stiffness of neck. The next day weakness and numbness of the left leg ensued. On admission to a local hospital his temperature was 102°, pulse 98, and respirations 22. He showed signs of meningeal irritation with paresis and hyporeflexia of the left leg. WBC count 15,700; CSF contained 220 WBC. He was transferred to Blodgett Hospital with a tentative diagnosis of acute poliomyelitis.

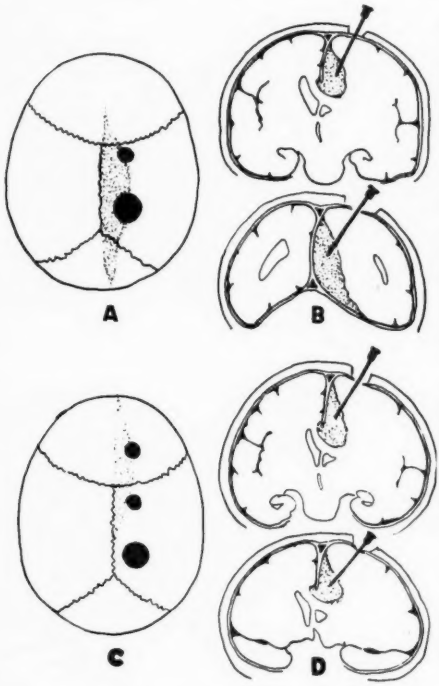

FIG. 1. Schematic drawings of right interhemispheral subdural empyema. (A, B) First operation. (C, D) Second operation. A and C, skull viewed from above to show trephine openings and suppuration (stippled). B and D, cross sections of head showing evacuation of pus.

Examination. The patient was acutely ill and lethargic. Temperature 101.8°, pulse 58 and respirations 18. B.P. 120/78. There was mucopurulent discharge from the nose and congestion of turbinates. His neck was moderately stiff; Brudzinski and Kernig signs were positive. Paresis and hyporeflexia of left lower extremity were noted with distal diminution of sensation. Babinski sign was positive on the left. Laboratory studies: Urine contained albumin but no casts. RBC 3,790,000; WBC 17,700: 5 non-segmented forms, 77 segmented and 17 lymphos. CSF contained 807 WBC: 91 per cent polys. and 9 per cent lymphos.; total protein 150 mg., sugar 74 mg., but culture and smear negative. Roentgenograms revealed clouding of right frontal, ethmoid and maxillary sinuses. *Tentative diagnosis:* Right pansinusitis with intracranial extension by thrombophlebitic route and with meningeal reaction. The patient was given 100,000 units of penicillin and 0.3 gm. streptomycin every 3 hours intramuscularly.

Further Course. The day after admission a series of left Jacksonian convulsions occurred, affecting mostly the leg. Temperature had risen to 104.8°, pulse 80, and respirations 24. Drowsiness and signs of meningeal irritation had increased. The eyegrounds remained normal but there was complete left homonymous hemianopsia. The left hemiparesis had become practically complete in the leg, moderately severe in the arm, but only minimal in the face. Superficial and deep sensations were disturbed on the left side of the body, particularly in the leg. *Diagnosis:* Acute right subdural empyema, presumably in a posterior and parasagittal (interhemispheral) localization.

Operation. On the same day (Mar. 24, 1937), under local anesthesia, a posterior parietal trephine opening was made close to the midline. After opening the dura, no subdural suppuration or meningitis was found on the convexity but the brain was under increased pressure. When an exploring cannula was introduced towards the

interhemispheral fissure (touching the falx), pus gushed out under pressure. A second trephine opening was then made in the anterior parietal parasagittal area, and here again pus was obtained from the interhemispheral fissure (Fig. 1 A & B). A total of 40 cc. were evacuated in this manner; 2 cc. of thorotrast were then injected into the posterior trephine hole into the interhemispheral fissure. Subsequent roentgenograms showed the interhemispheral space and falx faintly outlined by the contrast medium, most of which had escaped into the dressing.

The posterior incision was reopened and the bony defect enlarged. After removal of a small amount of brain tissue, the interhemispheral fissure was exposed for more adequate drainage. This attempt, however, failed because of high intracranial pressure and hence it was impossible to introduce penicillin into the obliterated interhemispheral space. The wounds were therefore closed.

Culture of Pus. Hemolytic Staphylococcus aureus, coagulose positive.

Course. Systemic penicillin and streptomycin therapy was continued and also anticonvulsant drugs were given. His condition improved; the convulsions stopped, and temperature dropped to 101°, yet hemiparesis and headaches persisted. Three days later, his condition deteriorated again, with a rise of temperature to 103.8°, pulse 120, respirations 22. The convulsions recurred, affecting this time mostly the left arm and the left side of the face. It was thought that this indicated spreading of the subdural empyema anteriorly.

Operation. On Mar. 27, 1947, under local anesthesia, the two parietal trephine holes were reopened and another trephine opening was made in the parasagittal posterior frontal region (Fig. 1 C & D). Tapping of the interhemispheral fissure through this new frontal burr hole evacuated 15 cc. of pus, and also aspiration of the anterior parietal opening yielded pus, but none was obtained from the posterior parietal trephine hole. Two small rubber catheters were placed into the interhemispheral fissure, one through the frontal, the other through the anterior parietal trephine opening, and 50,000 units of penicillin instilled into each catheter. The wounds were closed in a single layer, leaving the catheters in place.

Course. For the next 3 days 40,000 units of penicillin were injected daily through the catheters, in addition to the systemic antibiotic therapy. After a stormy convalescence the patient improved slowly, his fever subsided, the hemiparesis cleared up, and he was eventually discharged home.

He was seen on several occasions in May and June 1947. He had become asymptomatic except for persistent dull headaches. Neurologic findings were normal, but the WBC count remained elevated. However, early in July 1947 (12 weeks after his original illness) he began to complain of increasingly severe right frontal headaches with occasional vomiting. This led to his readmission on July 15, 1947.

Second Admission

He was acutely ill, suffering from severe right frontal headache. Temperature 100.6°, pulse 80, and respirations 16. The posterior parietal decompressive area was bulging. There was right temporal tenderness and congestion of scalp veins. Signs of meningeal irritation were absent. The eyegrounds were normal but the left homonymous hemianopsia had recurred. He past-pointed with the left arm to the left and Babinski sign was positive on the left. *Diagnosis:* Residual right occipital brain abscess.

Course. He was immediately placed on a regime of 100,000 units of penicillin intramuscularly every 3 hours. Subsequent ventriculography (July 16, 1947) showed

considerable displacement and deformation of the moderately enlarged ventricular system, characteristic of a right occipital mass lesion (Fig. 2 A & B). The ventricular fluid contained 15 polys.

Operation. On the same day, under combined local and intravenous sodium pentothal anesthesia, a small right parieto-occipital osteoplastic flap was turned. An irregular mass of granulation tissue with multiple areas of inspissated pus was

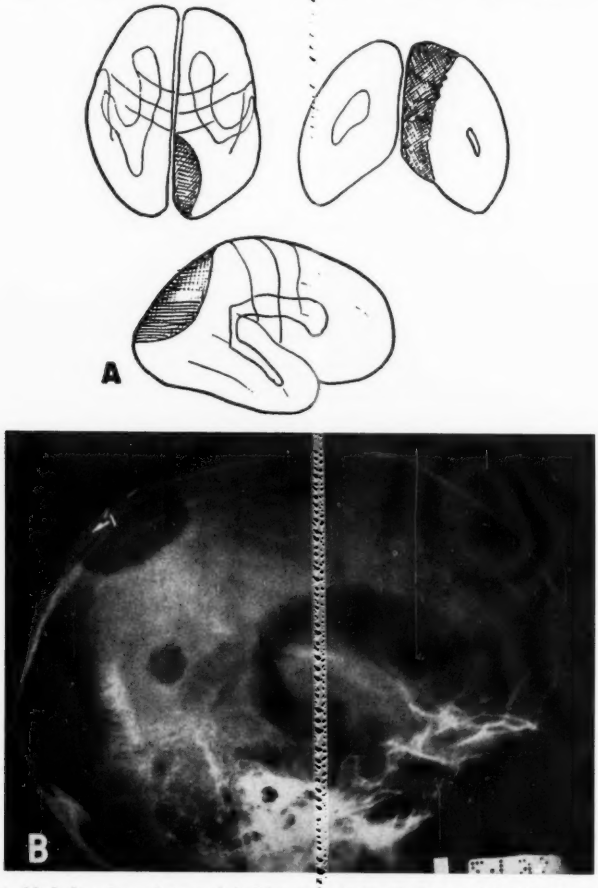

FIG. 2. Right-sided chronic parieto-occipital interhemispheral abscess. (A) Line drawings showing ventricular deformity and lesion (resected). (B) Left lateral ventriculogram.

removed from the interhemispheral fissure of the occipital region, together with adjacent scarred and degenerated brain tissue (Fig. 2 A & B). After instillation of 100,000 units of penicillin into the operative cavity, the wound was tightly closed without drainage.

Histologic Diagnosis of Tissue Removed. Subchronic abscess with marked perivascular inflammatory changes in the brain tissue.

Further Course. After an uneventful postoperative course of 6 days, severe headaches developed, temperature rose to 101°, and there was bulging of the wound. Purulent CSF began to drain from the incision but under continued systemic penicillin and streptomycin therapy, and occasional intraventricular injections of penicillin (10,000 to 20,000 units per injection), the wound gradually healed and the patient became afebrile. On discharge (Aug. 23, 1947) he was asymptomatic except for complete left homonymous hemianopsia.

He felt quite well until early September 1947, when he again experienced increasingly severe right temporal headache.

Third Admission

On readmission (Sept. 9, 1947) he suffered from excruciating paroxysms of right retro-orbital pain associated with nausea and vomiting. Temperature was 101.4°. His neck was stiff but Kernig sign negative. The eyegrounds were normal but the left hemianopsia was still present. WBC count 11,100 with 67 segmented, 18 non-segmented, 2 monos, and 13 lymphos. CSF was under a pressure of 220 mm. and contained 175 WBC: 75 per cent polys. and 25 per cent lymphos.; total protein 55 mg. Culture and smear were sterile.

Course. Penicillin was again given systemically, 60,000 units every 3 hours. It was felt that he had a residual epitentorial abscess under the basal surface of the right temporo-occipital lobe. This diagnosis was confirmed by ventriculography, which indicated a mass encroaching on the generally dilated ventricular system from the right tentorial surface (Fig. 3 A & B).

Operation. On Sept. 14, 1947, under local anesthesia, supplemented by intravenous sodium nembutal, the right parieto-occipital flap was reopened and an additional small right temporo-occipital flap elevated. The swollen brain contained a walnut-sized cortical encapsulated abscess in the right temporo-occipital lobe. This abscess was adherent to the tentorium and continued as an irregular mass of subdural granulation tissue with inspissated pockets of pus on the upper surface of the tentorium to the incisura. The entire inflammatory mass was removed by block dissection, but was broken and, unfortunately, some pus spilled. Adjacent fibrotic and degenerated brain tissue of the temporo-occipital lobe was resected. A small opening accidentally made into the enlarged posterior horn of the right lateral ventricle was immediately sealed by gelfoam. The dura was not closed. Penicillin (50,000 units) was injected into the large cavity produced by the partial resection of the occipital lobe. The scalp was closed in two layers, after replacing the bone flaps.

Culture of Pus from Abscess. Non-hemolytic Staphylococcus aureus (coagulose negative).

Histologic Diagnosis of Specimen. Granulation tissue with multiple abscesses and marked degenerative and inflammatory changes of the surrounding brain.

Further Course. The patient was given 0.25 gm. of streptomycin and 100,000 units of penicillin intramuscularly every 3 hours. His course was febrile with signs of mild meningeal irritation. Because the lateral ventricle had been accidentally opened, repeated intraventricular penicillin injections, 30,000 units each, were given. The ventricular fluid contained from 50 to 175 cells (75 per cent polys.) but remained sterile. Eventually the meningeal irritation subsided. The wound healed well and the patient's general condition was improved by repeated blood transfusions. He was discharged on Oct. 8, 1947 without symptoms except for the residual left hemianopsia.

Although he felt entirely well, he was readmitted for a final check-up on Mar. 9, 1948.

Fourth Admission

He had been able to return to school in the interval period. Neurologic findings were normal except for the unchanged complete left hemianopsia. The scalp over the trephine openings was sunken in. Temperature and WBC count were normal.

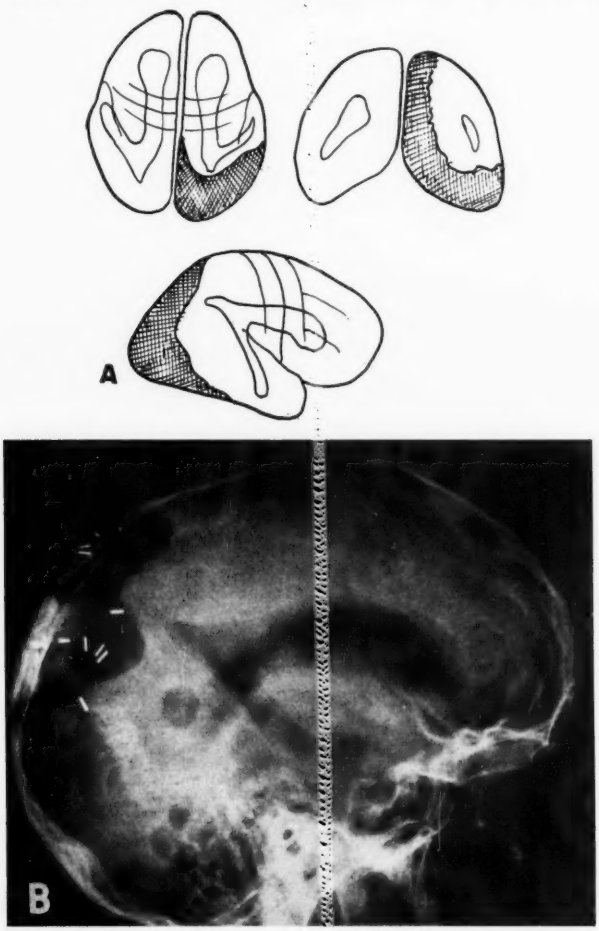

FIG. 3. Chronic right temporo-occipital epitentorial abscess. (A) Line drawings showing ventricular deformity and lesion (resected). (B) Left lateral ventriculogram.

Ventriculography (Fig. 4 A, B & C) revealed generalized ventricular dilatation with a greater widening of the right occipital and temporal horns. There was (postoperative) occipital porencephaly and shift of the ventricular system to the operative opening on the right, in short, the picture of an atrophic temporo-occipital encephalopathy. The ventricular fluid was normal in all respects.

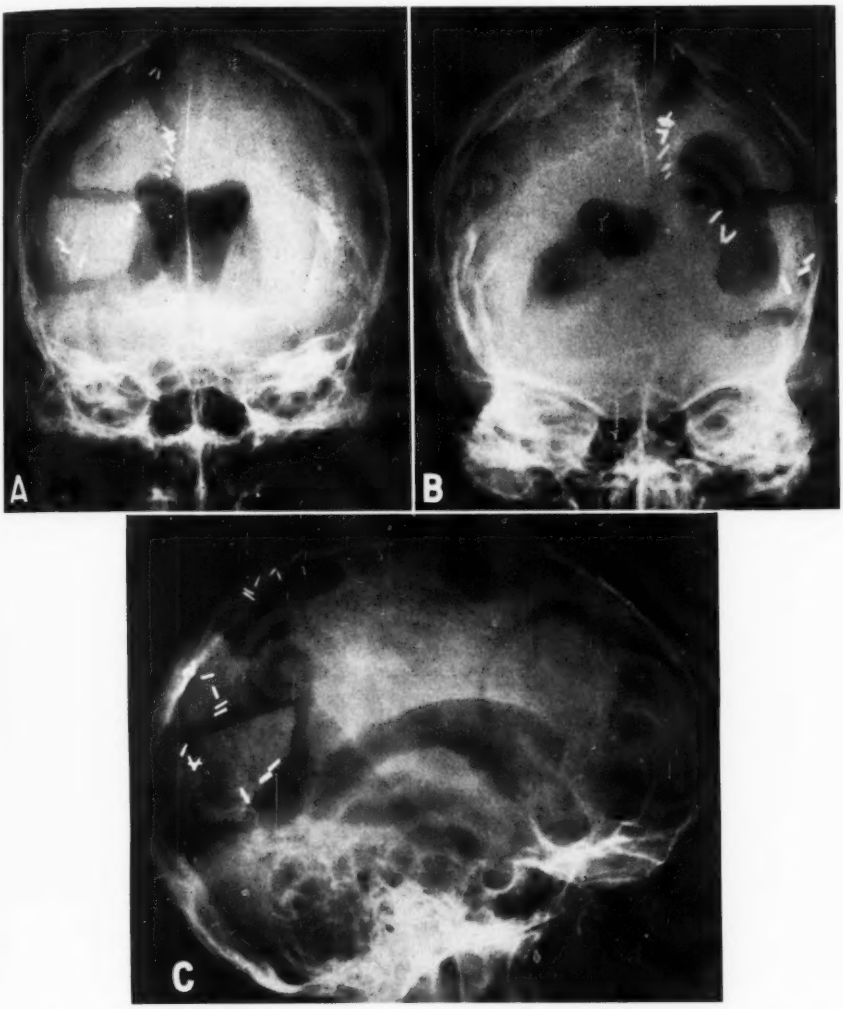

FIG. 4. Postoperative ventriculograms after recovery from right hemispheral and epitentorial subdural abscess. (A) AP projection. (B) PA projection. (C) Left lateral projection. Ventricular system is generally dilated and shows traction deformity and porencephalic widening of posterior portion of right lateral ventricle. Note faint impregnation of falx with residual thorotrast which had been previously injected into the interhemispheral empyema.

When last heard of, 2 years and 3 months after his final operation, the patient was in good health and working. He has had no convulsive attacks.

PATHOGENESIS

In order to understand the clinical symptomatology, the pathogenesis of acute and chronic subdural suppuration must be briefly reviewed.

Acute pyogenic infection of the subdural space develops either by direct or indirect route. In the first case, a neighboring suppurative process extends directly through the dura. Purulent frontal sinusitis, e. g., may lead to osteomyelitis of the posterior wall of the frontal sinus with necrosis of bone, formation of an extradural abscess and finally, to subdural empyema with or without actual perforation of the dura. The indirect vascular route of infection is probably more common: In severe frontal sinusitis, septic thrombophlebitis of mucosal or orbital veins develops and continues into diploë of the skull, dural veins and sinuses, and finally, into cerebral veins. The exposed course of some of the dural and cerebral veins within the subdural space favors the formation of subdural empyema. Once the infection has reached the subdural space it spreads rapidly over the cerebral convexity without meeting obstacles. It is apt to invade the interhemispheral fissure and to extend from there around the falx to the other side, especially when the purulent process originates at the orbital surface of the frontal pole. With the bedridden patient in recumbent position, the subdural exudate gravitates to the posterior part of the cerebral hemisphere, particularly to the angular space formed by the falx and tentorium. Thus, acute subdural empyema develops progressively in an anterior-posterior direction affecting either the dorsolateral surface of the hemispheral convexity, or the interhemispheral fissure, or both regions.

Chronic subdural suppuration was practically unknown before the antibiotic era because, in the past, the patients almost never survived the acute stage. When an acute subdural empyema is inadequately drained or surgery delayed too long after administration of large doses of antibiotics, the process becomes chronic. The originally liquid pus thickens, and begins to organize and loculate. It tends to disappear from the surface of the hemispheral convexity and to retreat into the hidden recesses of the interhemispheral fissure and the contiguous epitentorial space. Typical chronic subdural abscess forms an irregular mass of pyogenic granulation tissue with tongue-like extensions into cerebral sulci containing multiple small pockets of inspissated pus. It may be associated with clusters of cortical brain abscesses.

CLINICAL SYMPTOMATOLOGY AND DIAGNOSIS

It is of great practical importance not only to diagnose the presence of subdural empyema, but also to determine its location. This is frequently possible by clinical evidence alone, despite the diffusely spreading character of the suppuration.

Empyema of the frontoparietal convexity and Sylvian fissure causes focal convulsive attacks involving eyes, face, and upper extremity. This is soon followed by a faciobrachial type of hemiparesis and aphasia (in left-sided lesions). As the hemiparesis increases, there is usually paralysis of conjugate gaze to the paretic side.

On the other hand, interhemispheral empyema produces the following

clinical picture: The initial Jacksonian attacks affect the leg. Hereafter, paresis and sensory loss occur in the lower extremity and last, severe hemiplegia develops with sparing of facial muscles. Homonymous hemianopsia is another even more significant diagnostic sign in such cases. Visual field defects and sensory changes may be accurately demonstrated at an early stage, but when the patient's consciousness becomes progressively clouded their presence may be difficult to show.

Chronic subdural abscess must be suspected when a patient, after apparent recovery from acute subdural empyema, complains of headaches and when there is evidence of persistent latent infection (fever, leucocytosis, and pleocytosis of the spinal fluid). There may be also recrudescence of preexisting neurologic signs, such as the recurrence of visual field defects in the reported case.

Trephination is the best method to confirm the clinical diagnosis of acute subdural empyema. It permits verification of the lesion, identification of the causative organism, and, at the same time, surgical drainage of pus. Neither ventriculography nor angiography is advisable in the acute stage. In suspected interhemispheral empyema, the trephine opening(s) must be made close to the midline. Even though no gross changes of the exposed cortex may be evident, an exploring cannula is introduced towards the interhemispheral fissure until it strikes the falx. Pus is then obtained as a rule. In the past, the situation was frequently misinterpreted: The surgeon who tried to find the suspected abscess through a trephine opening placed somewhere at the convexity eventually encountered pus at a great depth. It was then assumed that a deep intracerebral abscess had been tapped, but not realized that the pus was located in the interhemispheral or epitentorial space. Injection of a small amount of thorotrast into the empyema will eliminate any doubt about the localization. As shown by Schiller, Cairns and Russell,[7] and also Keith and McKenzie,[4] the contrast medium diffuses in a characteristic fashion in the interhemispheral and epitentorial space. Even after drainage, minimal residuals of phagocytized thorotrast may permit roentgenographic visualization of falx and tentorium (Fig. 4A & B).

In chronic subdural abscess both ventriculography or angiography are helpful for diagnosis and safe to use, but may not always permit differentiation of subdural abscess from intracerebral abscess. As our case shows, both conditions may co-exist.

TREATMENT

Acute subdural empyema must be regarded as an absolute emergency. Two objectives must be obtained: First, control of infection and secondly, removal of pus. Ordinarily it is considered a sound principle to identify the responsible organism before selecting the appropriate drug, but in subdural empyema no time must be wasted by awaiting the outcome of bacteriologic studies. Antibiotics and sulfonamides are immediately given in massive doses before surgery is attempted. At least 100,000 units of penicillin should

be injected every 3 hours either intravenously or intramuscularly, and, if necessary, combined with 0.3 gm. of streptomycin intramuscularly and sulfadiazine in doses of 6 gm. in 24 hours. Even though antibiotics and sulfonamides inhibit the spread of infection so that the body gains time to mobilize its reactive forces, it would be a serious error to rely entirely on drugs and to procrastinate surgically. The extremely grave condition of most patients demands immediate operation. The pus is liquid and easily drained in the early stage, but prolonged treatment with antibiotics transforms it into a thick plastic exudate which is difficult to remove.

In acute interhemispheral empyema the primary trephine opening should be placed over the suspected site of the lesion, as close as possible to the midline. If pus is found, additional burr openings are made along the midline and above the transverse sinus. Three to four parasagittal trephine openings and one or two openings above the transverse sinus are adequate. Thorough *primary* evacuation of the interhemispheral and epitentorial pus is essential. In the case reported here, the vast extent of the suppuration was at first not recognized. Consequently, secondary drainage became necessary. A residual loculation of pus in the angle between the falx and tentorium was overlooked, resulting in the formation of a chronic subdural abscess. The subdural space is more easily drained after collapsing the cerebral hemisphere by lumbar puncture. This procedure, suggested by Kahn,[3] greatly facilitates the evacuation of empyema of the convexity, but it is less effective in interhemispheral empyema because the bridging veins prevent wide separation of the brain from the falx and tentorium. After the pus has been thoroughly removed by aspiration and subsequent irrigation with Ringer's solution, a soft rubber catheter is introduced through each of the burr holes into the interhemispheral or epitentorial space and left in place. Penicillin or streptomycin are injected through each of the inlying catheters, 2 or 3 times daily for several days. Even though systemically administered penicillin will readily enter the subdural space, a much higher and sustained local concentration of the drug is attained by topical instillation. In addition, some drainage of purulent exudate may be accomplished through the catheters for approximately 24 to 48 hours. Schiller, Cairns and Russell[7] inject into the catheters every 3 hours for several days large quantities of penicillin solution containing only 500 units of penicillin per cc. They feel that the quantity of irrigating fluid is more important than the concentration of the drug and that this method is more likely to prevent loculation of the empyema. Gurdjian and Webster,[2] and the author, instill about 30,000 to 50,000 units of penicillin in a smaller volume of fluid 3 times daily. The criticism may be voiced that large doses of concentrated penicillin solution in contact with the cortex may produce convulsions, particularly in patients who, due to thrombosis of cortical veins, have become more susceptible to convulsive attacks; yet the danger of convulsions appears a lesser evil than a spread of infection. Seizures, therefore, must be accepted as a calculated risk which can be reduced by anticonvulsant medication.

After 3 to 5 days, the inlying catheters may be removed while systemic treatment with antibiotics and sulfanamides is continued.

Keith and McKenzie[4] recently advocated removal of a long narrow median or paramedian strip of bone for more effective drainage of the midline fissure. This procedure, which was successfully used in 3 of their cases, has disadvantages. It increases the magnitude and risk of the operation by uncovering a long segment of superior longitudinal sinus. Furthermore, it deprives the sinus of its bony protection without necessarily assuring better drainage than can be obtained by multiple trephine holes.

Because of the difficulties of accomplishing adequate primary drainage, interhemispheral empyema may become chronic. If that condition is suspected, ventriculography or angiography should be employed for accurate diagnosis. Should these studies confirm the presence of a chronic interhemispheral abscess, an osteoplastic flap may be safely turned over it because, by this time, the infection is well walled off and local immunity sufficiently high to prevent new dissemination by surgical procedures. It is, of course, necessary to maintain a high blood level of antibiotics and sulfonamides before and after operation. Granulomatous areas must be completely excised, if possible by block dissection without breaking into the multiple abscesses. Degenerated and scarred brain tissue may have to be sacrificed in order to remove potential epileptogenic foci. There is little objection against such procedures in the frontal and occipital areas and at the basilar surface of the temporal lobe (although permanent hemianopsia may ensue). In the Rolandic area surgery must, by necessity, remain more conservative in order to avoid hemiplegia. A lesson learned in the reported case was to expose not only the interhemispheral region, but also the epitentorial space. At the first craniotomy only the interhemispheral parieto-occipital portion of the abscess was removed but the epitentorial extension overlooked. The remaining part of the abscess flared up again and required a second craniotomy. Accidental opening of the ventricle or spilling of purulent material must be avoided, but should these undesirable events happen, the danger of serious wound contamination is slight. Topical application of antibiotics, combined with intraventricular and general systemic administration will, as a rule, prevent complications. Tight closure of the wound without drainage is desirable and safe in chronic cases.

Before the patient is regarded cured, pneumography should be made for a final check-up. In recovered cases, air studies give the appearance of an atrophic brain lesion with enlargment and traction deformity of the lateral ventricles towards the site of the lesion. The subarachnoid spaces may be partially obliterated.

SUMMARY

Acute subdural empyema occurs more frequently in the interhemispheral fissure and epitentorial space than was formerly realized. Interhemispheral suppuration produces a characteristic clinical syndrome consisting of initial

Jacksonian attacks of the opposite leg followed by motor sensory hemiparesis, predominantly of the lower extremity, and homonymous hemianopsia. The diagnosis is verified by parasagittal trephination with aspiration of pus. Roentgenographic visualization of the empyema by injection of thorotrast permits differentiation from intracerebral abscess.

The treatment consists of immediate massive administration of antibiotics (penicillin, and/or streptomycin) and sulfonamides. This is to be followed soon by surgical removal of the pus. Adequate primary drainage is essential and can be obtained through multiple parasagittal and epitentorial trephine openings. Subsequently, the subdural space is intermittently irrigated through inlying catheters with antibiotic solutions. The interhemispheral and epitentorial spaces also are preferential sites of chronic subdural abscess. This condition develops from inadequately treated acute subdural empyema. The symptomatology of chronic subdural abscess is similar to that of chronic intracerebral abscess. Ventriculography or angiography are usually necessary for diagnosis. The treatment of chronic subdural abscess requires osteoplastic craniotomy with complete excision of the abscess and of the granulomatous areas. Definitive cure of the patient should be verified by postoperative pneumography.

An illustrative case is reported in detail.

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VARIATIONS IN THE TRIFURCATION OF THE SEMILUNAR GANGLION AND SURGICAL IMPLICATIONS

HARVEY GASS, M.D., AND WILLIAM P. VAN WAGENEN, M.D.
Department of Surgery, Neurosurgical Division, Strong Memorial Hospital, and The University of Rochester School of Medicine and Dentistry, Rochester, N. Y.

(Received for publication November 10, 1949)

OCCASIONALLY, after performing what one considers to be an adequate section of the posterior sensory root of the trigeminal ganglion for tic douloureux by the temporal route, he is surprised that the postoperative examination shows the area of anesthesia on the face to be considerably less than was anticipated.

In 1932 van Nouhuys,[3] on the basis of anatomical dissections, concluded that "the sensory root of the fifth nerve is not composed of the three parts that correspond to the three peripheral branches from the gasserian ganglion" and that because of the interlacing ramifications and anastomoses within it, partial section of the sensory root "cannot be regarded as an absolutely reliable procedure."

The interlacing fibers within the posterior root had been noted by others[1,2] yet, aware of that, Spiller and Frazier[4] took issue with van Nouhuys, insisting that despite such interlacings, on purely clinical evidence from their own vast experience with this operation, the conclusion was indisputable that the supply of the mandibular division was from the outer portion of the root; of the maxillary division, from the middle portion of the root; and of the ophthalmic division, from the inner portion. In view of the general satisfaction of most neurosurgeons since then with partial section of the posterior root transtemporally, the conclusions of Spiller and Frazier seem justified. Nevertheless, others[5] have been impressed with occasional atypical areas of skin anesthesia following operations for which van Nouhuys' observations, if pertinent, may not offer the complete solution. The possibility occurred to us that variations in the topography of the semilunar ganglion, its roots and branches, not appreciated by the surgeon in his limited surgical exposure, were responsible for such results. This matter has prompted the following investigation.

The points at which the semilunar ganglion branches into its three divisions anteriorly (Fig. 1, a and b) are definitive in determining the relationship of the posterior sensory root and the ganglion to commonly used surgical landmarks. If the trifurcation of the ganglion takes place far anteriorly (pre-fixed ganglion), the surgical anatomy would be somewhat different than if it occurs relatively far posteriorly (post-fixed ganglion). In the latter instance, a surgeon could, for example, in orienting himself from the foramen spinosum, readily mistake the fibers of the mandibular division

for those of the ganglion itself or even for those of the entire posterior sensory root. Accordingly, the purpose of this study was to determine by actual measurements whether such significant variations actually do occur.

Measurements were made in 16 fresh anatomical specimens of the semilunar ganglion of each side *in situ*. Particular attention was paid to the re-

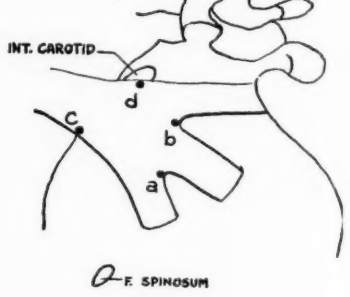

FIG. 1

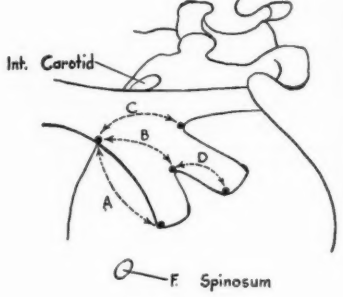

FIG. 2

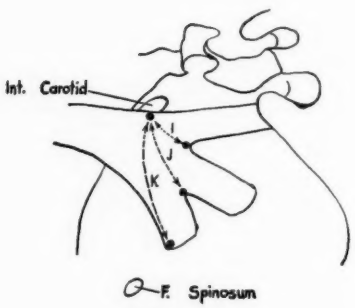

FIG. 3

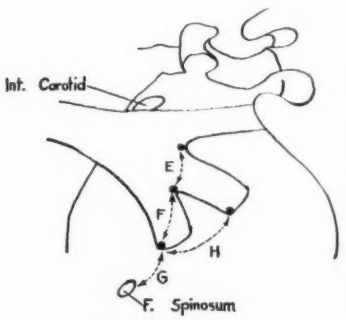

FIG. 4.

lationship of the points of trifurcation (Fig. 1, a and b) to certain anatomical landmarks. Certain other dimensions were taken also. In each dissection the overlying dura and dura propria of the ganglion were stripped off sufficiently to visualize the borders of the roots and ganglion clearly without disturbing their anatomical setting.

The posterior edge of the petrous ridge at the groove for the trigeminal nerve (Fig. 1, c) and the foramina ovale and rotundum were used as anatomical reference points. From these points measurements were made to determine variations of trifurcation in an anteroposterior direction. Four such measurements were made: A, B, C, and D (Fig. 2).

In a transverse direction variations in the location of points of trifurca-

tion were determined by 3 measurements: I, J and K (Fig. 3). Reference points for these measurements were the foramen ovale and the medial border of the ganglion at a point opposite to the internal carotid as it ascends from the foramen lacerum (Fig. 1, d). The latter point is not an anatomical constant because of the possible variations in the tortuosity of the artery. Such variations, however, were noted to be slight and not usual.

FIG. 5. Semilunar ganglion and roots drawn to actual size of average dimensions.

Certain other dimensions of the ganglion and its parts were also measured. These were distances E, F, G, and H (Fig. 4).

Fig. 5 shows the actual average size of the ganglion and roots.

All measurements involving the foramina were uniformly made at the lateral anterior edge of each foramen.

RESULTS

In the anteroposterior direction distance A (Fig. 2) varied from 16 to 26 mm., B from 13 to 20 mm., C from 14 to 22 mm., and D from 9 to 19 mm.

Anteroposterior variations in the location of the points of trifurcation in different specimens can perhaps be more easily visualized by considering the ratio of B to A and C to A. B/A varied from 0.63 to 1.00 whereas C/A varied from 0.62 to 1.18. The average value for B/A for the entire series was 0.778. The average value for C/A for the entire series was 0.821.

Analysis of these ratios by sex revealed that in this series females were more apt to have a pre-fixed ganglion, with trifurcation taking place an-

teriorly, whereas males were more apt to have post-fixed ganglions, with posteriorly placed trifurcations. The average value of B/A for females was 0.807 compared to 0.761 for males and the average value for C/A in females was 0.857 compared to 0.799 in males.

Analysis by age failed to reveal any significant correlation between age and type of fixation of the ganglion.

In the transverse direction the dimension K varied from 16 to 24 mm., J, from 9 to 16 mm., and I, from 3 to 9 mm. Variations in the transverse direction, therefore, were somewhat less than those in the anteroposterior direction.

The width of the roots at their origin (E and F, Fig. 4) showed only slight variation. E and F each varied only from 6 to 10 mm.

Considerable variation was found to exist in the separation of the foramina. Thus distance G (Fig. 4) was found to range from 4 to 13 mm. Moreover, the foramen spinosum in particular showed considerable variation in its size in different specimens, being in one instance hardly larger than the size of a pin-point and in another as large as the eraser on a pencil.

It is noteworthy that while measurements on one side of a specimen were usually matched by measurements of a similar magnitude on the opposite side, this was not invariably so. Substantial differences were rarely present between the two sides, especially in the anteroposterior direction.

SUMMARY

Significant variations in dimensions of the semilunar ganglion and its roots, of as much as 1 cm., especially in the anteroposterior direction, have been found in 32 fresh postmortem dissections. The points where the ganglion trifurcates into its roots may thus be relatively far forward in one instance or far posterior in another. In women, it appears that one might expect anteriorly placed trifurcation more commonly, whereas in men a posterior trifurcation would be more apt to be the rule.

In the surgical exposure of the ganglion and its posterior root via the temporal approach, the surgeon frequently finds it difficult to obtain wide visualization of these structures. In view of the aforementioned variations, with a minimal operative exposure the operator subjects himself to possible errors in obtaining a good differential section of the posterior root. It is suggested, therefore, that before cutting what he takes to be the posterior root, the surgeon strive to obtain visualization of these structures for not less than 1 cm. in the anteroposterior direction unless the points of trifurcation of the ganglion are otherwise clearly in view for his orientation. If this is not feasible and there is doubt as to exact orientation, it might probably be wise to make his incision through the fibers in a slanting direction posteriorly so as to get behind the trifurcation of the ganglion.

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UNILATERAL PREFRONTAL LOBOTOMY FOR THE RELIEF OF INTRACTABLE PAIN

REPORT OF 58 CASES WITH SPECIAL CONSIDERATION OF FAILURES*

JOHN E. SCARFF, M.D.

Department of Neurological Surgery, College of Physicians and Surgeons, Columbia University, and the services of Neurological Surgery, Neurological Institute, Columbia-Presbyterian Medical Center, New York, and the Veterans Administration Hospital, Bronx, New York

(Received for publication November 12, 1949)

UNILATERAL prefrontal lobotomy for the relief of intractable pain has now been performed by me or my associates at the Neurological Institute in 58 consecutive cases.[1,2,3] In every instance the lobotomy was performed under direct vision in the plane determined by the coronal suture and the sphenoid ridge, passing just anterior to the tip of the lateral ventricle.[3] There was no mortality and little morbidity despite the fact that many of these patients were bad surgical risks.

Results have been classified as good, fair and poor. A result has been considered good if the patient, after operation, no longer voluntarily complained of pain nor required narcotics; fair, when the patient, after operation, still complained of some pain but was comfortable with far less narcotic than before operation; poor, when the patient experienced no lessening of pain and there was no reduction in narcotics required after the lobotomy.

Good results were obtained in 38 of the 58 patients, representing 66 per cent of the total series. In approximately two-thirds of these cases the pain was due to inoperable carcinomas, including those of the mouth, neck and cervical spine. The follow-up periods on these cases averaged 5 months (Table 1).

TABLE 1

Pain—good results

No.	Patient	Pathology	Side of Pain	Pain Relieved on			Dominant or Non-dominant	Follow-up (months)
				Ipsi-lateral	Contra-lateral	Bi-lateral		
1	Aaronson	Cranial metastases from pelvic carcinoma	bilateral			bil.	N-Dom.	3 died
2	Abosch	Carcinoma of tongue	right	ips.			N-Dom.	3 died
3	Rivera	Carcinoma of tongue	left	ips.			Dom.	2 died
4	H.D.	Carcinoma of jaw	left	ips.			Dom.	1½ died
5	W.B.	Carcinoma of jaw	right		cont.		Dom.	3½ died
6	Bishop	Trigeminal neuralgia	left	ips.			Dom.	3 died

* Read before the Fourth International Neurological Congress, September 9, 1949, Paris.

TABLE 1—*continued*

No.	Patient	Pathology	Side of Pain	Pain Relieved on			Dominant or Non-dominant	Follow-up (months)
				Ipsi-lateral	Contra-lateral	Bi-lateral		
7	Jewett	Atypical facial pain	right	ips.			N-Dom.	17
8	Calarino	Atypical facial pain	left	ips.			Dom.	17
9	Larson	Thoracic aneurysm	left	ips.			Dom.	10
10	Redfield	Carcinoma of pancreas	left	ips.			Dom.	6 died
11	Johns	Carcinoma of pancreas	right	ips.			N-Dom.	9 days died
12	Turner	Neuritis, lumbosacral	bilateral			bil.	Dom.	1
13	Grass	Carcinoma of uterus	left	ips.			Dom.	5 died
14	Fajardo	Carcinoma of bladder	left	ips.			Dom.	4 died
15	Miner	Metastasizing carcinoma of humerus	right	ips.			N-Dom.	4 died
16	Wilds	Metastasis to spine (cervical, thoracic, lumbar) and femur	bilateral			bil.	N-Dom.	5 died
17	Blumberg	Compression fracture, T-12; Parkinsonism	right		cont.		Dom.	3
18	Bozzo	Metastasizing carcinoma, T-9	bilateral			bil.	Dom.	1½ died
19	Stiner	Metastases, both ilia, ischia, femora	bilateral			bil.	Dom.	1 died
20	S. Rosalie	"Arthritis, spine"	bilateral			bil.	Dom.	9
21	Greenberg	Arthritis, cervical	right	ips.			N-Dom.	9
22	Irwin	Neuritis, cauda equina	bilateral			bil.	N-Dom.	9
23	Schlichting	Carcinoma of pancreas	bilateral			bil.	Dom.	½ died
24	Behrens	Carcinoma of cervix	right	ips.			N-Dom.	7½
25	Perry	Adhesive arachnoiditis	bilateral			bil.	Dom.	7
26	Gainsford	Carcinoma of bladder	bilateral			bil.	Dom.	5
27	Washington	Carcinoma of large bowel	bilateral			bil.	N-Dom.	5
28	Rickard	Carcinomatosis of pelvis	bilateral			bil.	N-Dom.	4½
29	Barletta	Carcinoma of tongue	right	ips.			N-Dom.	4
30	Wilson	Thalamic pain, post-apoplexy	left	ips.			Dom.	4
31	Patterson	Carcinoma of pancreas; metastases to liver	bilateral			bil.	N-Dom.	1 died
32	Wuench	Spinal cord varicosities	bilateral			bil.	N-Dom.	3
33	Van Keuren	Carcinoma of cervix with extension	left	ips.			Dom.	2
34	Nason	Squamous cell carcinoma, ext. aud. meatus	left	ips.			Dom.	2
35	Murphy	Carcinoma of tongue with metastases	bilateral			bil.	N-Dom.	1 died
36	Stein	Carcinoma, base of skull and neck	bilateral			bil.	N-Dom.	9
37	Westlake	Carcinoma of rectum	left		cont.		N-Dom.	1
38	Speed	Carcinoma of breast, metastases to brachial plexus	left	ips.			Dom.	½

A fair result with partial relief of pain was obtained in 12 patients, or 20 per cent of the series. This group brings the number of patients materially benefited by the operation to 86 per cent of the total number (Table 2).

Poor results followed operation in 8 patients, or 14 per cent of the series (Table 3).

Detailed psychometric, psychiatric and social studies carried out on a large number of these patients before and after operation established the fact that no significant impairment of intellect or personality followed the unilateral lobotomy.[3] This fact makes unilateral lobotomy applicable in a variety of situations where bilateral lobotomy is not.

TABLE 2

Pain—fair results

No.	Patient	Pathology	Side of Pain	Pain Relieved on			Dominant or Non-dominant	Follow-up (months)
				Ipsi-lateral	Contra-lateral	Bi-lateral		
1	G.B.	Phantom limb syndrome	left	ips.			Dom.	14
2	Allen	Superior sulcus tumor of lung	left	ips.			Dom.	6
3	King	Carcinoma of jaw with fractured mandible	left	ips.			Dom.	4 died
4	J.C.	Hemicephalalgia, idiopathic, greatest in face	left	ips.			Dom.	5
5	Remling	Carcinoma of pancreas	bilateral			bil.	Dom.	5 died
6	Devine	Metastasizing carcinoma, C7, T1 from breast	right	ips.			N-Dom.	2
7	J.Y.	Palindromic rheumatism	bilateral			bil.	Dom.	10
8	C.C.	Chronic myositis	bilateral			bil.	Dom.	7
9	W.K.	Urethra, psychogenic	bilateral			bil.	Dom.	7
10	Bassoff	Tabes dorsalis	bilateral			bil.	N-Dom.	4
11	Coker	Carcinoma of pancreas, metastasis to liver	bilateral			bil.	N-Dom.	2
12	Ishoo	Carcinoma of rectum	bilateral			bil.	N-Dom.	1*

* Bilateral lobotomy performed later. See Table 4.

Termination of drug addiction due to pain was greatly facilitated by unilateral lobotomy. All narcotics, it was found, could be abruptly stopped following operation without any of the customary symptoms or signs of withdrawal.[3]

The mechanism whereby pain was relieved by unilateral prefrontal lobotomy is not understood.[1] In cases of unilateral pain the results were equally good whether the lobotomy was performed on the side of the brain ipsilateral to the somatic pain or contralateral to it; in cases of bilateral pain the results were equally good whether lobotomy was performed on the dominant or the non-dominant hemisphere (Table 1).

My earlier reports on unilateral lobotomy for the relief of pain have dealt primarily with statistical analysis of good, fair and poor results, evaluation of

effects on intellect and personality, and description of the facilitation in the termination of drug addiction.[1,2,3] This present report deals primarily with an analysis of failures.

The group of 8 patients in my series who experienced no relief of pain following unilateral prefrontal lobotomy included 7 with advanced carcinoma (Table 3). These tumors were not essentially different in character or location from those in patients who experienced good results. It appears

TABLE 3

Pain—poor results

No.	Patient	Pathology	Side of Pain	Side Operated on			Dominant or Non-dominant	Result
				Ipsi-lateral	Contra-lateral	Bi-lateral		
1	Pensata	Schmirke tumor, base of skull; involvement Gasserian ganglion	left		cont.		N-Dom.	No relief*
2	Heine	Carcinoma of uterus; extension to lumbosacral plexus	right		cont.		Dom.	Relief 4 weeks; recurrence*
3	Hellweg	Carcinoma of bronchus; metastases to brachial plexus	right	ips.			N-Dom.	Relief 4 weeks; died 2 months
4	Auchincloss	Carcinoma of breast; metastases to cervical spine and brachial plexus	right	ips.			N-Dom.	Relief 6 weeks; died 4 months*
5	Ryttenberg	Carcinoma of breast; multiple metastases to cervical, thoracic and lumbosacral spine, and to pelvis and long bones	bilateral			bil.	N-Dom.	No relief
6	Radich	Carcinoma of bronchus; metastases to brachial plexus	right	ips.			N-Dom.	Relief 4 weeks; recurrence
7	Silhan	Carcinoma of pelvis; extension to lumbosacral plexus	left		cont.		N-Dom.	No relief*
8	Koenderman	Tabes dorsalis	bilateral			bil.	N-Dom.	No relief

* Bilateral lobotomy performed later. See Table 4.

significant, however, that in these cases with poor results the tumors impinged directly upon large sensory nerves, either cranial nerves or major elements of the brachial or lumbosacral plexuses. It is reasonable, therefore, to believe that the pain produced by these tumors was of maximum intensity. This is the only factor that appeared to distinguish the cases with poor results from those with good results.

It may be concluded from this that *unilateral prefrontal lobotomy interposes a relative, rather than an absolute, barrier between pain reception and*

pain perception at the conscious level; that this barrier is completely effective for many degrees of pain; but that it may be only partially effective for pain of maximum intensity.

If this concept be accepted, then many of the other facts regarding unilateral prefrontal lobotomy become ordered and understandable. For in-

TABLE 4

Bilateral lobotomy

Patient	Pathology	Side of Pain	Unilateral Lobotomy	Interval Between Uni. and Bil. Lobotomy	Bilateral Lobotomy	Follow-up	Result After Bilateral Lobotomy
1. Pensata	Schminke tumor, base of skull; involvement Gasserian ganglion	left	right (N-Dom)	4 mos.	left (Dom)	3 mos.	Poor Pain intolerable; unrelieved by drugs.
2. Heine	Carcinoma of uterus; extension to lumbosacral plexus	right	left (Dom)	5 mos.	right (N-Dom)	2 mos. died	Fair Patient ambulatory to day of death; reasonably comfortable on codeine .030 gm. q4h.
3. Auchincloss	Carcinoma of breast; metastases to cervical spine and brachial plexus	right	right (N-Dom)	3 mos.	left (Dom)	2 mos. died	Fair 4 wks. before death required Dilaudid .002 gm q2h; 2 wks. before death, morphine .010 gm. q2h.
4. Silhan	Carcinoma of pelvis; extension to lumbosacral plexus	left	right (N-Dom)	1 mo.	left (Dom)	7 mos.	Good Cheerful, ambulatory, but does require aspirin and codeine .030 gm. tab. occasionally.
5. Ishoo*	Carcinoma of rectum	bil	right (N-Dom)	2 wks.	left (Dom)	1 mo.	Good No voluntary complaints of pain, no analgesics prescribed; however, under pentothal narcosis he complained bitterly of pain.

* This patient had experienced a fair result from unilateral lobotomy. However, he is a native of Iran, allowed into this country for 3 months. In view of the advancing malignancy and his imminent need to return to Iran, bilateral lobotomy was performed.

stance, in the group of patients with partial relief of pain following lobotomy, there were 6 with inoperable carcinoma (Table 2—Cases 2, 3, 5, 6, 11, 12). The partial failure in these cases can be ascribed to the same mechanism as total failure in the group just cited, total and partial failures differing only in the matter of degree.

It has been said that the effectiveness of unilateral lobotomy in the relief of pain is of short life. It is undoubtedly true that a small number of patients experience only temporary relief after operation. But in attempting to interpret this fact, it should be kept clearly in mind that unilateral

lobotomy represents only a relative barrier to pain perception and not an absolute one; and that if the pain increases sufficiently in intensity, it may penetrate that barrier. In my own series there were 4 who experienced relief of pain following lobotomy only to suffer recurrence of pain 4 to 6 weeks later (Table 3—Cases 2, 3, 4, 6). It is important to note that each of these patients was in the last stages of a rapidly progressing malignancy and that the return of pain coincided with advances in the disease. It is my own opinion that return of conscious pain in these cases was because the intensity of the painful stimuli increased to a point which permitted penetration of the relative barrier, not because the barrier itself became less. It is significant that the return of pain occurred in only 5 patients of the entire series; also, that in no instance was there a late return of pain when the clinical course of the disease indicated that the severity of the painful stimuli had remained constant.

Bilateral lobotomy was performed eventually in 5 patients who had been unrelieved of pain by unilateral lobotomy (Table 4). Two of these realized complete and permanent relief; 2 experienced relief not quite complete; and 1 gained no relief at all. It appears, therefore, that bilateral lobotomy, although somewhat more effective than the unilateral operation, also represents a relative, rather than an absolute, barrier to conscious pain perception.

The personality of the patient appears to influence to a considerable degree the success or failure of unilateral lobotomy in relieving pain. In the group of 12 patients who continued to complain of some pain after lobotomy there were 5 who did not have carcinomas, nor, in fact, any organic lesion that could be anatomically verified (Table 2). These were diagnosed as phantom limb syndrome (Table 2—Case 1), atypical facial pain (Table 2—Case 4), chronic arthritis without x-ray changes (Table 2—Case 7), chronic myositis (Table 2—Case 8) and pruritis of the vulva, present since childhood (Table 2—Case 9). All of the usual methods of investigation and local treatment of such conditions had been exhausted before the patients came to me. Psychoneurotic or pre-psychotic stigmata were clearly recognizable in each of them.

After unilateral lobotomy these 5 patients appeared definitely better to the objective observer; their faces revealed no suffering, they had good appetites, they slept well, they carried out full daily routines of activity. But whenever they saw a doctor or nurse, they would invariably complain of pain and ask for medicine. They frequently claimed that the pain was even greater after the operation than it had been before—a statement never made to me by any of those patients suffering from carcinoma. Placebos or hypodermic injections of sterile water usually allayed their complaints.

My limited experience with these 5 patients indicates that where there exists within an individual a psychogenic need for pain unilateral prefrontal lobotomy will not eradicate that need nor terminate the pain. Neurotics, psychoneurotics and psychotics, therefore, offer poor prognosis.

SUMMARY

Unilateral prefrontal lobotomy performed for the relief of pain in 58 consecutive cases yielded good results in 66 per cent, fair results in 20 per cent, and poor results in 14 per cent. This operation produces a relative, rather than an absolute, barrier to conscious pain perception. Experience indicates that the effectiveness of unilateral lobotomy is permanent for pain of constant severity. Pain of psychogenic origin has not been relieved by unilateral lobotomy.

I wish to thank Dr. Lester A. Mount of the Neurological Institute for permission to include in this report two patients operated upon by him: Case 24, Behrens, and Case 25, Perry; also Dr. Edward Schlesinger of the Neurological Institute for his kind permission to include his patient, Case 36, Stein, in this report.

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THE SURGERY OF EPILEPSY

LIMITATIONS OF THE CONCEPT OF THE CORTICO-ELECTROGRAPHIC "SPIKE" AS AN INDEX OF THE EPILEPTOGENIC FOCUS*

RUSSELL MEYERS, M.D., JOHN R. KNOTT, PH.D., ROBERT A. HAYNE, M.D., AND DONALD B. SWEENEY, M.D.

Division of Neurosurgery and Department of Psychiatry, State University of Iowa College of Medicine, Iowa City, Iowa

(Received for publication November 18, 1949)

MOST neurosurgical procedures directed against epilepsy have been fashioned upon the generally accepted hypothesis that a hyperirritable cortical focus ("epileptogenic focus," "firing point") is a prime factor in the pathogenesis of epileptic seizures. The hypothesis itself is based upon the pioneer work of Hughlings Jackson (1861–83), Fritsch and Hitzig (1870), Ferrier (1873), Munk (1880), Gowers (1881) and Talbert (1899), and from it follows logically the concept that the alleviation of epileptic seizures may be expected from (a) identification of the hyperirritable focus and (b) its extirpation.

Unfortunately, the number of individuals who have obtained complete relief from epileptic attacks following surgical procedures does not conform closely to theoretic expectations. Even when grossly apparent lesions, such as cortico-meningeal scars, cysts of the cortex, and circumscribed atrophic areas are encountered at operation, only about one-fifth of patients subjected to operation can be freed of their seizures (Penfield and Erickson[6]). When, on the other hand, grossly apparent lesions are not in evidence and the surgeon is under the necessity of demonstrating the hypothetical firing point by physiologic means only, the results thus far have proven similarly disappointing.

This disparity between expected and actual clinical results is commonly accounted for by (a) citing the various practical difficulties encountered in identifying the cortical firing point; (b) assuming incomplete extirpation of the epileptogenic focus; (c) supposing the existence of more than one firing point; and/or (d) citing the inadvisability in certain instances of extirpating the cortical focus because of the likelihood of impairing crucial functions, such as speech and eupraxia. The fundamental concept of the cortical epileptogenic focus is, of course implicitly contained in all four of these accounts.

In consideration of the first three explanations (a, b and c above) the realization of better results would appear to reside in the improvement of methods for locating and precisely defining the limits of the cortical firing point. To this end, clinicians have striven toward more accurate observation and description of the convulsive seizure peculiar to each patient. Improved types of cortical stimulators have been designed, affording increasing control

* Presented at the meeting of the Harvey Cushing Society, June 2, 1949, New Haven, Connecticut.

over voltage, amperage, wave form, frequency and pulse-duration for threshold determinations of cortical tissue. Roentgenography, including pneumoencephalography and cerebral angiography, has been extensively utilized and during the past 15 years electroencephalography has been employed to the same end. In connection with the latter diagnostic procedure, the concept has been evolved that the cortico-electrographic "spike" may be considered an indicator of the epileptogenous focus.

The assessment of spikes in the electroencephalograms of patients with convulsive disorders has been the particular object of several recent investigations (Jasper,[2] Walker *et al.*,[8,9] Penfield and Jasper[7]). The general viewpoint relative to post-traumatic epilepsy has been summarized by Walker *et al.*:[9] "In post-traumatic epilepsy focal *spikey** waves or spikes about the site of the injury indicate a convulsive diathesis." In an extensive series of cases, Walker *et al.*[9] showed that areas of after-discharge of spike and associated phenomena could be localized electrocortically following infraliminal sine-wave stimulation. Of these findings they say:

"These observations seem to indicate that certain cortical areas adjacent to cerebral scars may be excited by infraliminal stimuli producing a state of local epileptic hypersynchrony. Although these phenomena have many characteristics of cortical after-discharge, their lower threshold, more localized activity and longer duration suggest that they are different, at least in degree. *That areas capable of such activity are potentially epileptogenic seems highly probable. That they are the foci, from which spontaneous epileptic attacks originate, lacks demonstration, but it seems a reasonable inference in those instances in which the electrocorticographic attack is associated with an aura identical to that in the spontaneous attacks. If the focus is some distance from the scar, another and perhaps primary focus may be located nearer the injury.*"†

Inferentially, then, the spike *per se* is regarded as evidence of an epileptogenic focus, and from this it would follow that excision of the spike zone may be expected to have a salutary effect on the patient's seizures.‡

Another view of "spikes" and "spikey" (or "sharp") waves is given by Jasper.[2] While he concurs in the notion that clean-cut spikes of brief duration are evidence of a focal zone of discharge, he considers that spike-like waves or "sharp" waves are the conducted end-result of spikes originating at a distance. The evidence supporting this view is convincing.

It is clear that caution must be exercised in differentiating between the two types of cortical potential change which may casually be included in the generic term "spiking." In this connection, Jasper and Droogleever-Fortuyn,[3] by stimulating midbrain and diencephalic structures, have demonstrated spikes formed with a dome simulating the familiar "petit mal" seizure pattern. These are, to be sure, not spikes in the sense that they are

* Italics ours.

† Italics ours.

‡ This inference was made rather explicit by Quadfasel and Walker[8] in a description of the delimitation of an epileptogenic focus. Their report was made relatively early after completion of an operative series and for this reason no statistical data were given. On the basis of clinical impression, however, the authors were inclined toward belief that the results were beneficial.

isolated. However, in some instances the dome can be absent, rhythmic spikes alone being apparent.

PROBLEM

The present investigation developed in the course of routine subcortical electrographic recording in a series of parkinsonian patients and non-parkinsonian "controls."[1,5] Among the latter were 4 epileptic patients, 1 of whom represented an instance of post-traumatic epilepsy. The routine EEG examination in this subject revealed frequent clear-cut spikes over the right frontal lobe—the chief site of cicatrization and gliosis following trauma. Following this, electrographic examination of the thalamic and striatal regions also revealed evidence of considerable spiking. The desirability of evaluating the incidence and behavior of cortical and subcortical spikes in epileptics thus suggested itself to us.

MATERIAL

Of the four epileptics examined, 1, a female of 23 years, failed to exhibit spikes (either cortical or subcortical) during the period of recording. The other 3 patients are reported upon. One of these was a 31-year-old male with post-traumatic epilepsy; another, a 10-year-old boy with a diagnosis of "idiopathic" epilepsy; and the third, a 36-year-old female with an "idiopathic" epilepsy since puberty and intractable pain of more recent origin in the left shoulder and arm due to Hodgkin's disease.

METHOD

Routine EEGs were recorded by means of 8 to 12 scalp leads and 2 ear leads. Both monopolar (scalp-ear) and bipolar (scalp-scalp) recordings were employed. "Depth" records were made by means of needle electrodes of the type developed by Gibbs and Gibbs. One of these needles had 8 pickup rings, the other 4. The former was regularly used for recording from the striatum; the latter, from the thalamus. Both monopolar (ring-ear) and bipolar (ring-ring) records were routinely obtained. The needles were placed in accord with methods previously described.[1,5] Immediately following electrographic recording in each patient, pneumoencephalography was carried out. By this means and the use of coronal brain sections cut on a grid, the approximate position of the ring leads was ascertained.[1,5]

The recordings of the epileptic subjects thus obtained were compared with recordings similarly derived from 25 "controls," 13 of whom were patients having no known organic brain disease and 12 of whom were suffering from extrapyramidal disorders. None of these 25 cases exhibited spikes in either scalp or "depth" recordings.

RESULTS

Case 1. Male, aged 31 years, post-traumatic epilepsy. The position of the electrodes is shown in Fig. 1. The major results are presented in Figs. 2 and 3, each a 10-second strip of record (all bipolar) from the frontal scalp, striatum and thalamus.

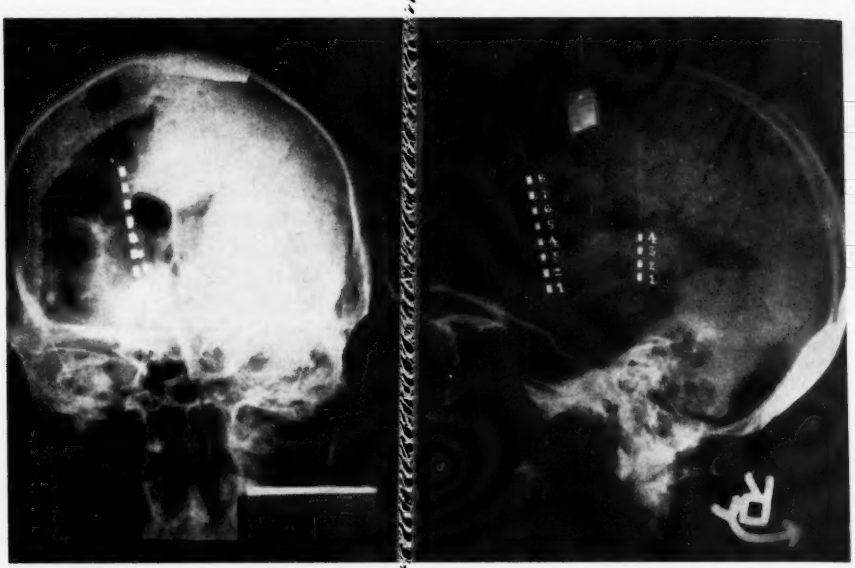

FIG. 1. *Case 1.* Male, age 31, post-traumatic epilepsy. Positions of striatal (8-ring) and thalamic (4-ring) electrodes as revealed by A-P and lateral views in pneumoencephalogram.

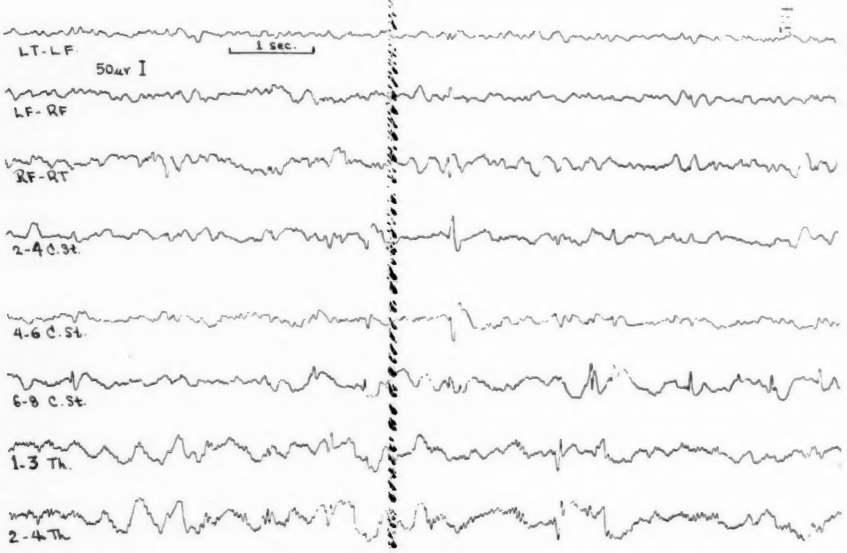

FIG. 2. *Case 1.* Male, age 31, post-traumatic epilepsy. Evidence of independent spike activity in corpus striatum and thalamus, bipolar recording. For positions of deep electrodes, see Fig. 1 and text.

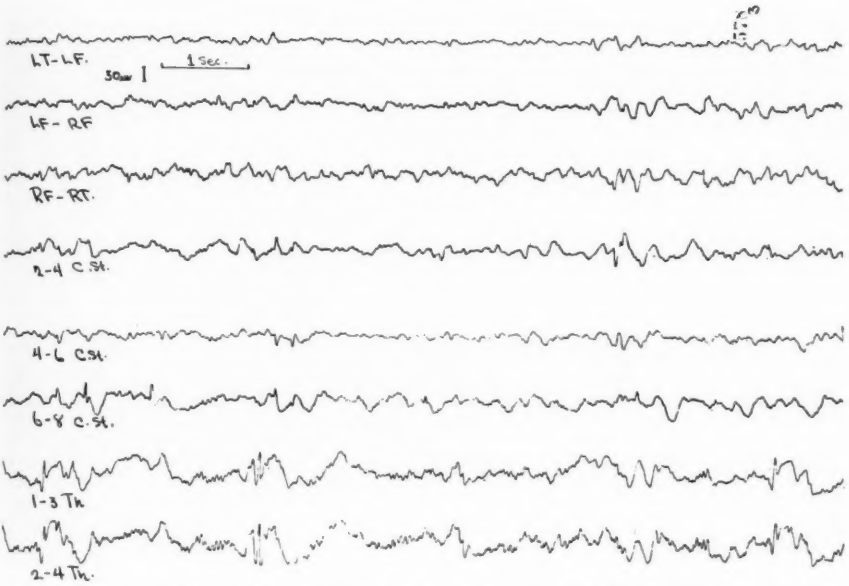

FIG. 3. *Case 1.* Male, age 31, post-traumatic epilepsy. Evidence of independent spike activity in thalamic leads, bipolar recording. For positions of deep electrodes, see Fig. 1.

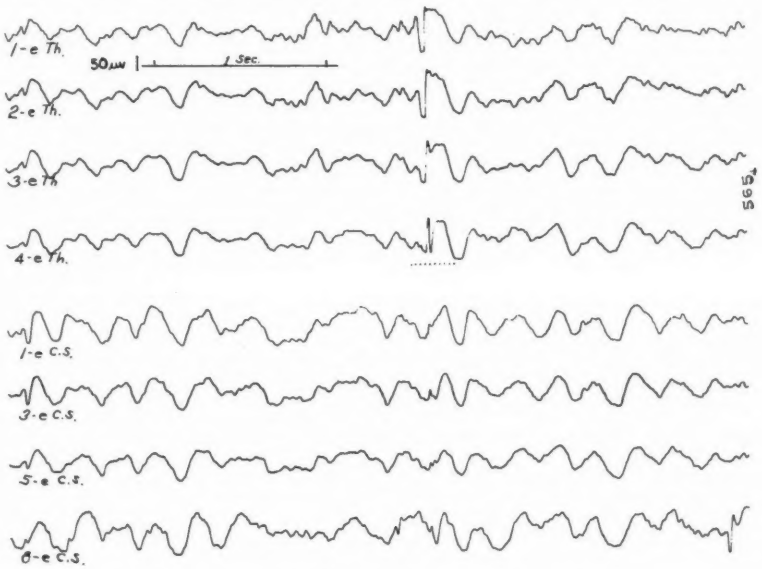

FIG. 4. *Case 1.* Male, age 31, post-traumatic epilepsy. Monopolar recording from thalamus and striatum, with greatest spike activity in Ring 1 of thalamus. For positions of deep electrodes, see Fig. 1.

In Fig. 2 there is evidence of *focal* spiking in the right frontotemporal area, in Ring 4 of the striatal lead, in Ring 6 of the striatal lead, in Ring 8 of the striatal lead and in Ring 3 of the thalamic lead. These foci are apparently quite independent. Fig. 3 shows further evidence of their independence, the scalp leads showing no clear instance of spiking, the thalamus a great deal, and the striatum a little spiking independent of that in the thalamus.

Monopolar deep leads are represented in Fig. 4. Here the thalamic leads are simultaneously active, the greatest amplitude apparently occurring in Ring 1 and the least in Ring 4. (All are negative with respect to the ear.) A little "spikey" activity appears in Rings 1, 3 and 8 of the striatal needle.

Case 2. Male, aged 10 years, "idiopathic" epilepsy. Evidence in Figs. 6 and 7 again shows that deeper structures may fire with a *spike-like* quality, independently

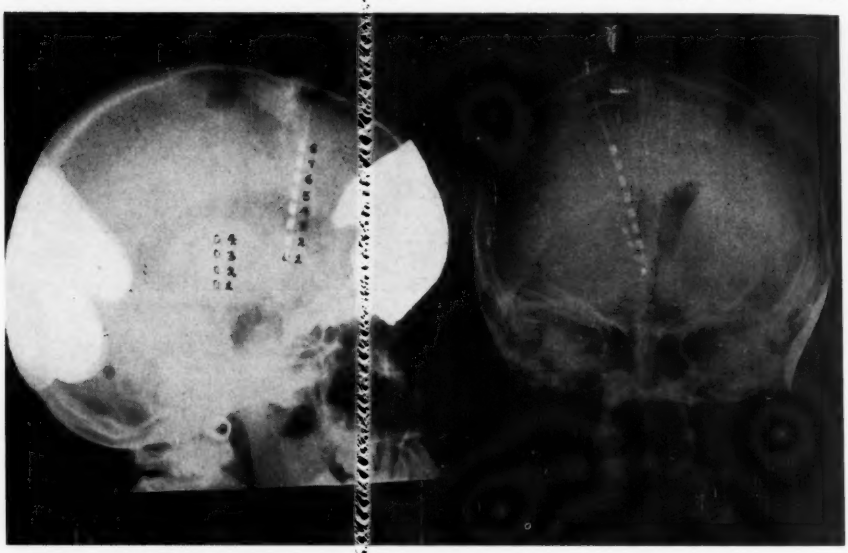

FIG. 5. *Case 2.* Male, age 10, "idiopathic" epilepsy. Positions of striatal (8-ring) and thalamic (4-ring) leads shown in A-P and lateral views in pneumoencephalogram.

of the surface, although in this case there is close following of certain striatal and thalamic leads. In Fig. 7, Rings 2–3 of the thalamic pickup show a considerable resemblance to Walker's published data on post-stimulation saw-tooth spikes. (Simultaneous EKG in this record rules out pulse artifact.)

Case 3. Female, aged 36 years, "idiopathic" epilepsy; Hodgkin's disease with intractable pain. In this patient, spikes were seen only with "activation" by intravenous sodium pentothal. The principal spike activity appeared only in striatal leads, referable chiefly to Ring 3 (Fig. 8). There was truncated spike-like activity, which may have been conducted by relay from a principal subcortical focus, apparent in the occipital lead; this was not at all times synchronized with the striatal spiking.

DISCUSSION

In a comprehensive discussion of the spread of epileptic activity in the cortex, McCulloch[4] has recently observed:

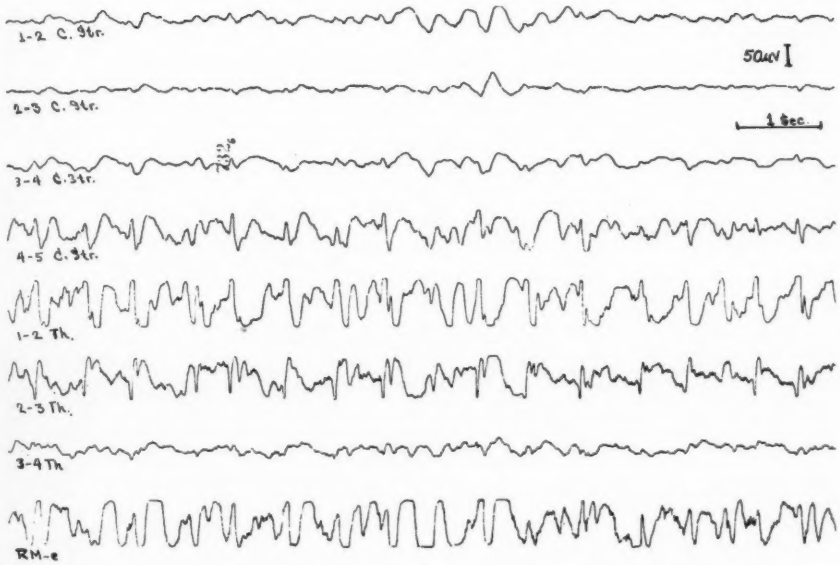

FIG. 6. *Case 2.* Male, age 10, "idiopathic" epilepsy. Evidence of "spikey" or spike-like activity in thalamic leads, bipolar recording. For positions of leads, see Fig. 5.

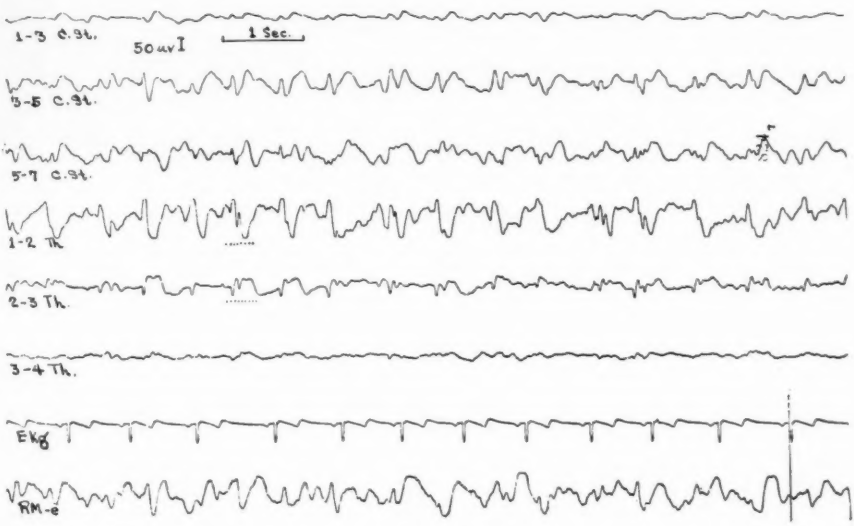

FIG. 7. *Case 2.* Male, age 10, "idiopathic" epilepsy. Evidence of thalamic "spikey" activity with striatal "following." For positions of electrodes, see Fig. 5.

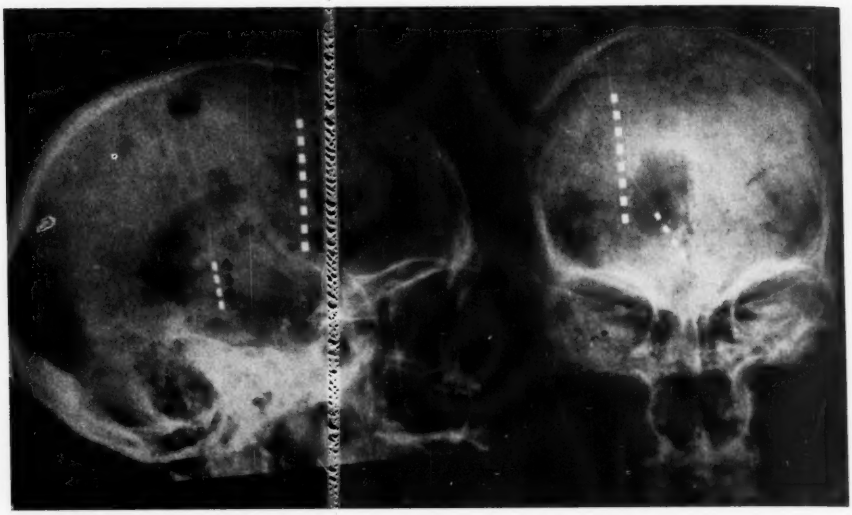

FIG. 8. *Case 3.* Female, age 36, "idiopathic" epilepsy, Hodgkin's disease with intractable pain. Positions of striatal (8-ring) and thalamic (4-ring) electrodes shown in A-P and lateral views in pneumoencephalogram.

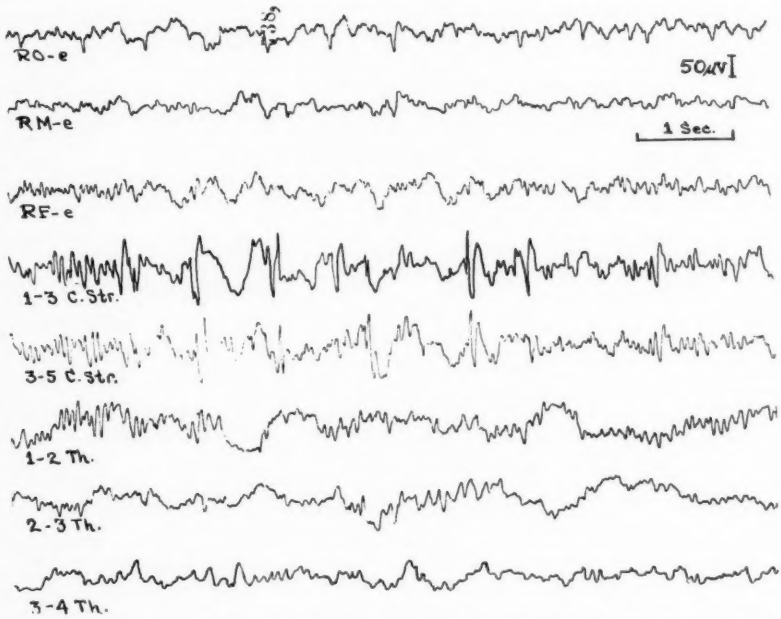

FIG. 9. *Case 3.* Female, age 36, "idiopathic" epilepsy, Hodgkin's disease with intractable pain. Evidence of spike activity restricted within corpus striatum, after "activation" with pentothal sodium (bipolar recording). For positions of leads, see Fig. 8.

". . . there is a . . . method of spread of seizures which we cannot ignore. It is probably more common than we expect and of late has begun to come to the fore. There are seizures . . . which may start in the cortex *or in other structures*,* but in which the seizures in the cortex play jack-in-the-box. You have them now at one place, and then they disappear out of the cortex, and then they reappear somewhere else in the cortex. . . . If one has enough electrodes in the lower structures, one sees it as a seizure which disappears out of the cortex but persists in some lower structures and then from them reappears in the cortex."

These comments are based on evidence (Ward, McCulloch and Kopeloff[10]) that demonstrated spread from an experimental cortical focus to other cortical areas and to the cerebellum (but, interestingly, not to the hypothalamus). These investigators assert that spread will be restricted to centers that receive axons or collaterals from the primary focus. After initiation of such activity in a remote region, the primary focus may die out, but further spread from the secondary area may occur, returning perhaps to the initial (primary) region. Wycis *et al.*[11] have also observed subcortical spiking, without concomitant cortical spikes, indicating that true subcortical foci, as we have described them, do exist.

We seem to have been in the box with "Jack," as well as outside, and so are in a position to make some comment about his unusual behavior.

In Case 1, there is every reason to believe that the seizures were on the basis of an epileptogenous zone in the vicinity of the demonstrable cicatrix. With its removal, clinical seizures (at least temporarily) have not recurred. Yet prior to operation, the ubiquitous "epileptogenic spike" appeared not only in this surface zone, but deeper in the cranial box. This deeper spiking was not restricted to one nuclear zone, but was distributed independently in at least two regions: the striatum and the thalamus. To analogize beyond McCulloch, the spike behaved like an ethereal Will-o'-the-wisp, its winking lamp leading us hither and yon, up and down: when we failed to see it, it may merely have been elsewhere, or it may not have been there at all.

In Case 2, the principal regions of spike-like activity were deep; scalp leads exhibited such "smoothing" as Jasper has attributed to conduction. (Actually, the raw data suggest smoothing in the sites studied, which would mean the primary focus might not have been tapped directly.)

Case 3 showed principally subcortical spiking, without primary cortical spiking, which, to use the analogies referred to above, suggests that Jack—or Jack's spring—was perhaps directly measured in his closed system.

Considering these cases together, spikes were derived from the thalamic and striatal leads in all 3 patients. They might appear during a particular time-interval in the thalamus alone, the striatum alone or in the surface leads alone. Combinations were also observed: thalamic spikes sometimes exhibited a "spread" to the striatum and/or cerebral surface, and in other instances striatal spikes sometimes exhibited a "spread" to the thalamus and/or cerebral surface. In still other instances, spikes apparently derived from the cerebral surface appeared to spread to the thalamic *or* striatal

* Italics ours.

region. In no case was a surface spike observed spreading to *both* the thalamic and striatal regions simultaneously.

CONCLUSIONS

The surgical implications would seem to be that spiking *per se* cannot be considered evidence of an epileptogenic focus of a primary sort.* Rather, the full statement of Walker must be used in the neurosurgical assessment of the electrographic spike activity: " . . . *spike about the site of the injury.*"† Yet, if subcortical spike genesis (Case 3, idiopathic epilepsy) is actually primary, "diathesis" may not always be consequent upon injury.

The present findings would seem to suggest that the concept of a cortical epileptogenic focus as the primary factor in the production of convulsive seizures stands in need of further evaluation. Even if this notion should ultimately prove tenable, we would still be under the necessity of critically inquiring whether the site of an epileptogenic focus is reliably revealed by the region(s) in which spike potentials happen to be recorded. Up to now, it has been tacitly assumed that this question is answerable in the affirmative. While the hypothesis has the appeal of engaging simplicity, there is a suggestion in the present data that "the epileptogenic firing point" is not invariably located in or just subjacent to the cerebral cortex, but that it may reside in deeper-lying nuclei, yet show corticipetal spread. This latter circumstance would explain the difficulty in identifying a cortical firing point responsible for convulsive seizures and account for the frequent failure of specific surgery directed against epilepsy.

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* This implication may also be drawn from the supporting evidence of the recent report of Wycis, Lee and Spiegel.[11]

† Italics ours.

INTERVERTEBRAL FORAMEN STUDIES

I. FORAMEN ENCROACHMENT ASSOCIATED WITH DISC HERNIATION*

LEE A. HADLEY, M.D.†
Syracuse, New York

(Received for publication November 18, 1949)

THESE studies are the outgrowth of observations coordinating the radiographic and morphologic studies of cadaver spines.[3] The specimens were supplied by the Anatomy Department at Syracuse University Medical College. Following radiographic examination the nerve roots were removed, embedded in paraffin and the section stained with hematoxylin eosin.

An examination of the normal lumbar foramen section (Fig. 1) shows the nerve

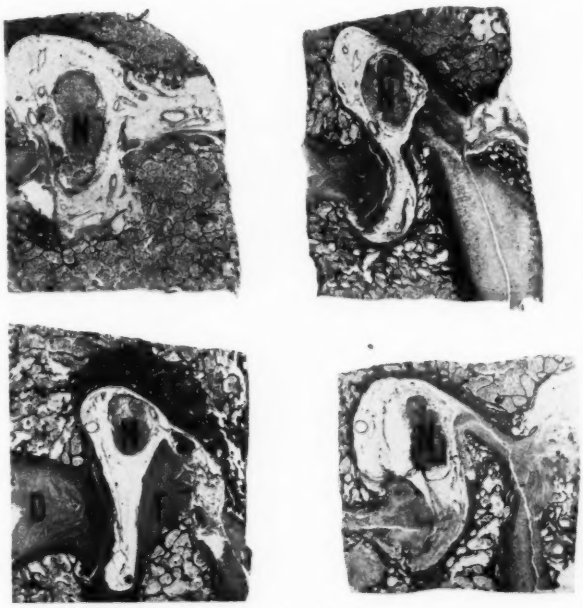

FIG. 1

root (N) or its ganglion occupying only about 1/6 to 1/4 of the opening. Surrounding the nerve is a generous reserve cushion space containing blood vessels, lymphatics,

* This work was aided by the Departments of Anatomy, Pathology and Photography, College of Medicine at Syracuse University.

† 925 State Tower Bldg., Syracuse, New York.

fat and areolar tissue. These compressible structures constitute a safety factor during the normal foramen constriction incidental to spinal movement. In each of the four sections here shown the disc (D) is on the left side. The ligamentum flavum (F) is somewhat hypertrophied in one of these sections.

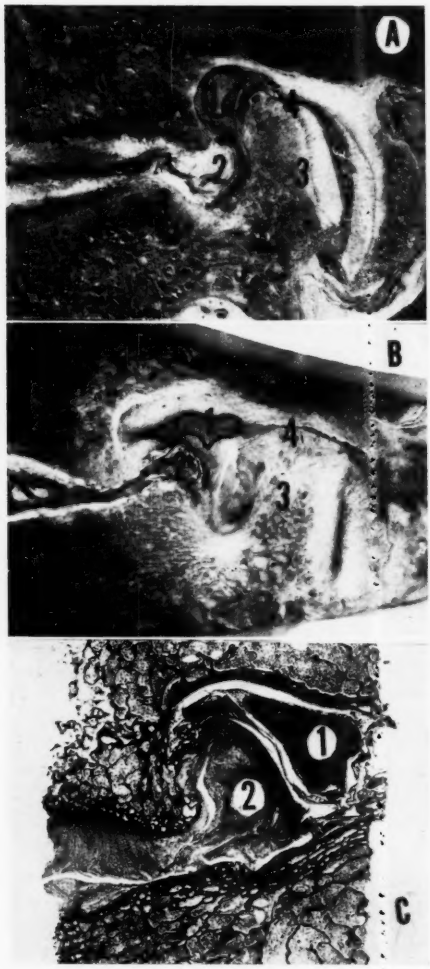

FIG. 2.

Permanent constriction of the foramen, from any cause, reduces the reserve cushion space. This predisposes to root pressure in the event of edema, hemorrhage, perineural inflammation or additional foramen encroachment.

The most common cause of foramen constriction is disc degeneration and its secondary changes. These include: herniation of disc substance into the foramen, thinning of the disc with approximation of the pedicles, subluxation of the upper vertebra forward or backward upon the one beneath, hyperplasia of the ligamentum flavum, bony spur formation projecting backward from the disc or forward from the posterior joint, and lastly, subluxation of the articular process from below upward and forward into the foramen. Some of these are illustrated in Fig. 2. A and B are blocks taken from constricted 5th lumbar foramina. In both, the disc is thinned and herniates backward (2) into the foramen. In these the distorted nerve (1) has been outlined. The superior articular process (3) from the sacrum has subluxated upward and forward into the foramen, constricting that opening. This has impinged against the under surface of the 5th pedicle at (4). Fig. 2C is the section (×1.7) from an encroached 5th lumbar foramen. The triangular-shaped nerve root (1) has become compressed between the pedicle above and the disc substance (2) herniating backward into the foramen.

Fig. 3 is the right lumbosacral spine of a male 83 years old. He had complained of low back pain for many years but no history of sciatica was obtained. A shows herniation of the 4th lumbar disc backward into the spinal canal (arrow) displacing the distal nerve roots. The

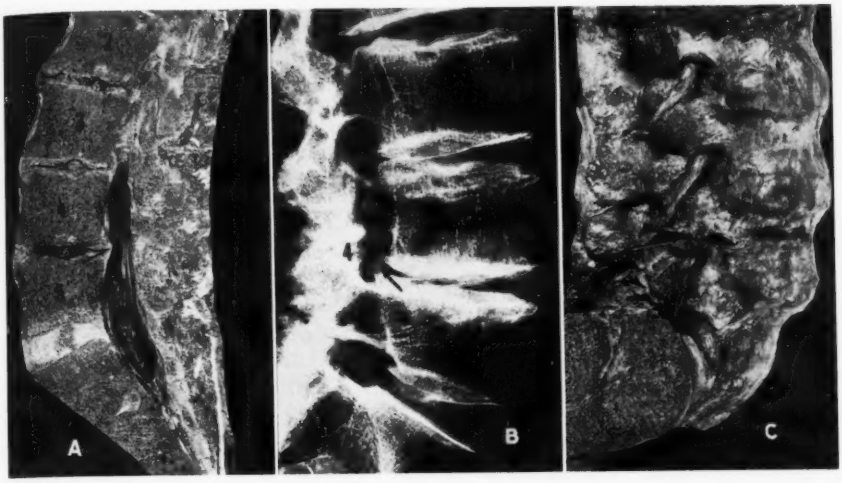

FIG. 3.

postmortem myelograph corroborated this disc protrusion and showed displacement of the distal roots. The lateral radiograph (B) indicates encroachment of the 4th intervertebral foramen (arrow). On the lateral photograph (C) the numerals indicate the posterior portion of each intervertebral disc. This man had a subluxation of the 4th and 5th posterior articulations with encroachment of the 4th foramen. Impingement of the 4th superior articular process against the under surface of the 3rd pedicle is indicated by the long arrow. The articulations and foramina above this point were normal. At the 5th posterior joint there is impingement of the sacral articular process

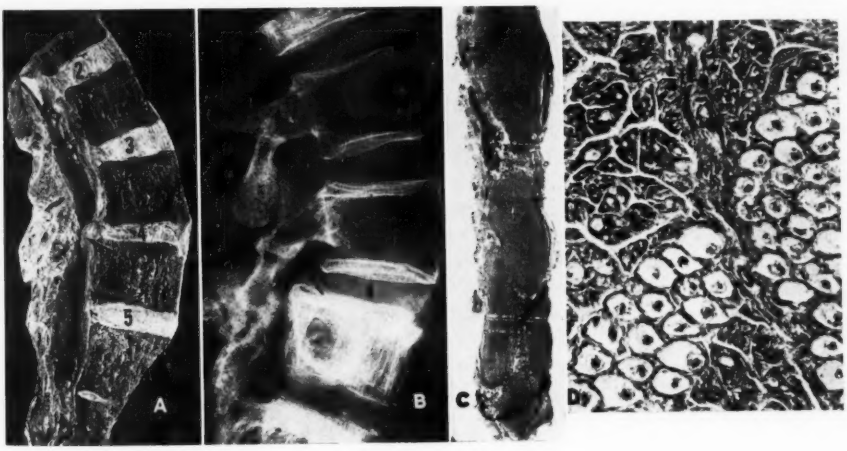

FIG. 4.

against the under surface of the 5th pedicle. A separate intra-articular ossicle, indicated by the arrowhead, has developed at this point.

Nerve roots 2, 3 and 5 appear normal and are surrounded within the foramen by a generous cushion space. Nerve root 4, however, is compressed and flattened by a decrease in the size of the foramen incidental to the posterior joint subluxation as well as a posterior herniation of the disc seen just above and behind the numeral 4.

A somewhat similar case was that of a male aged 62 who had complained of pain in the legs for 8 years before death. The sagittal section of the left side (Fig. 4A) shows a bulging backward of the 4th disc (arrow). The 5th lumbar nerve root was compressed against the posterior wall of the spinal canal at this point. The lateral radiograph (B) shows a spondylolisthesis of L4 forward on L5 but with no break in the neural arch. Marked constriction of the 4th foramen has occurred (arrow).

FIG. 5.

The ganglion was compressed by the encroached 4th foramen. Many of the large ganglion cells were flattened. Fig. 4C shows the ribbon-shaped 4th spinal nerve (×9) just distal to the ganglion. Marked flattening of the nerve and its constituent bundles has resulted from the herniation of disc substance into the foramen.

Fig. 4D is a section (×325) of the 5th lumbar root. This reveals evidence of nerve degeneration one space below the disc herniation. This high power section, distal to the ganglion, shows normal nerve fibers on the right side and in the lower left corner. Elsewhere there were patches of degenerative change, vacuolation and multiple nuclei in the same tubule. The 5th foramen was not encroached as seen in 4B. The herniated disc at L4 pressed against the 5th root. The patchy degenerative changes distal to the 5th ganglion here visualized may have resulted from that pressure.

These two cases illustrate how disc herniation within the canal may exert pressure upon a lower nerve root while contributing to encroachment of the adjacent foramen. The myelograph in each case would have shown a filling defect. In each the 4th foramen was encroached. Operative removal from the spinal canal of the 4th disc herniation, to relieve the 5th root pressure, would not have cured the foramen encroachment. Successful operative treatment would have necessitated, not only removal of the hernia for distal root pressure, but also proper attention to the adjacent foramen constriction.

Fig. 5 right side, same cadaver as Fig. 4, shows subluxation of the 4th posterior articulation with impingement of the articular process (arrow). The 4th foramen is constricted, while the 3rd and 5th foramina are normal and show ample reserve safety cushion spaces about the corresponding nerve roots. This photograph gives some indication of the difficult problem to be encountered in decompressing such a foramen by facetectomy.

Magnuson,[4] in discussing "the subject of the low back pain accompanied by so-called sciatica," states that the "approach has been mainly from the standpoint of root pressure from a ruptured intervertebral disc." He adds "this viewpoint seems much too narrow. It is quite apparent that variations in the path of the nerves are frequent not only in the foramen but at the exit from the foramen."

The importance of intervertebral foramen encroachment at once becomes apparent in properly evaluating nerve root pressure. At any operation to relieve intraspinal nerve root pressure, a very careful search within the intervertebral foramen even to its most lateral limits is always indicated. The surgeon must assure himself that no foramen encroachment exists.

The root compression may occur well lateral (Fig. 3C). The foramen is covered by elements of both articular processes. Various writers[1,2,5,6] have therefore recommended a complete facetectomy (removal of the entire posterior articulation) for decompression of the encroached intervertebral foramen, if that condition is present.

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INTERVERTEBRAL STUDIES

II. NERVE CHANGES INCIDENTAL TO FORAMEN ENCROACHMENT*

LEE A. HADLEY, M.D.†
Syracuse, New York

(Received for publication November 18, 1949)

FROM a study of the cadaver material[3] it was found that the compressed nerve may show alteration in its shape, fibrosis or certain morphological changes.

Fig. 1—Male, 85 years old. All nerve roots were surrounded by fibrotic changes and were adherent to the foramina.

1A and B are two corresponding sections of a bundle in the right 3rd lumbar nerve (×50) and taken a few mm. apart distal to the ganglion. In A the small nerve is seen arising from the parent nerve bundles (arrow) while in B it lies alongside and to the left of the nerve but outside the perineurium (arrow). Note that many of the Schwann tubules (small round white shadows) are empty in A but not in B.

1C is a high power (×450) study from the field indicated in 1A, and 1D is from the field in 1B. In 1C many of the Schwann tubules are empty and surrounded by large numbers of small nerve fibers. In 1D, same nerve bundle somewhat more distal, there are normal nerves, small fibers, and other tubules containing multiple nuclei. This latter is said to indicate a regenerative process.

Fig. 2—Same cadaver. Section from the left 5th root showing a nerve bundle surrounded by subperineural hemorrhage (arrows).

Fig. 3—Male, 75 years old. 3A shows impingement of the articular processes (arrows) and encroachment of the 5th lumbar foramen. There was compression and flattening of the nerve root. This structure was firmly attached to the foramen walls by a mass of dense fibrous tissue.

3B, a section (×12.5) of the left 5th lumbar root, removed by sharp dissection, is distal to the ganglion. It shows a concavity from pressure of the 5th intervertebral disc (arrow). Some of the nerve bundles are flattened and there is hyperplasia of the epineurium and perineurium at the upper portion of the root.

3C shows somewhat greater magnification (×75) of circular field indicated in 3B. Note hyperplasia of fibrous tissue. Some of the nerves have been destroyed, leaving empty Schwann tubules. A sclerotic intraneural arteriole is seen below and to the left of the center of the section (arrow).

A section of the 5th lumbar root from the right side of the same individual showed the nerve fibers normal but there was marked sclerosis of the intraneural arterioles. At least 12 were visualized within a single nerve bundle. The patient had complained of chronic pain in the right leg for some time before death.

A section taken from the 5th left motor root lying between the ganglion and the 5th disc showed a separation of nerve fibers and the endoneural spaces were enlarged. The appearance suggested edema of the endoneurium.

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* This work was aided by the Departments of Anatomy, Pathology and Photography, College of Medicine at Syracuse University.

† 925 State Tower Building, Syracuse, New York.

3D and 3E are both sections (×540) from the same left 5th lumbar nerve bundle but proximal to the ganglion and taken only a few mm. apart. The same arteriole (X) is noted to the left in each section. 3D shows nearly all of the nerves normal

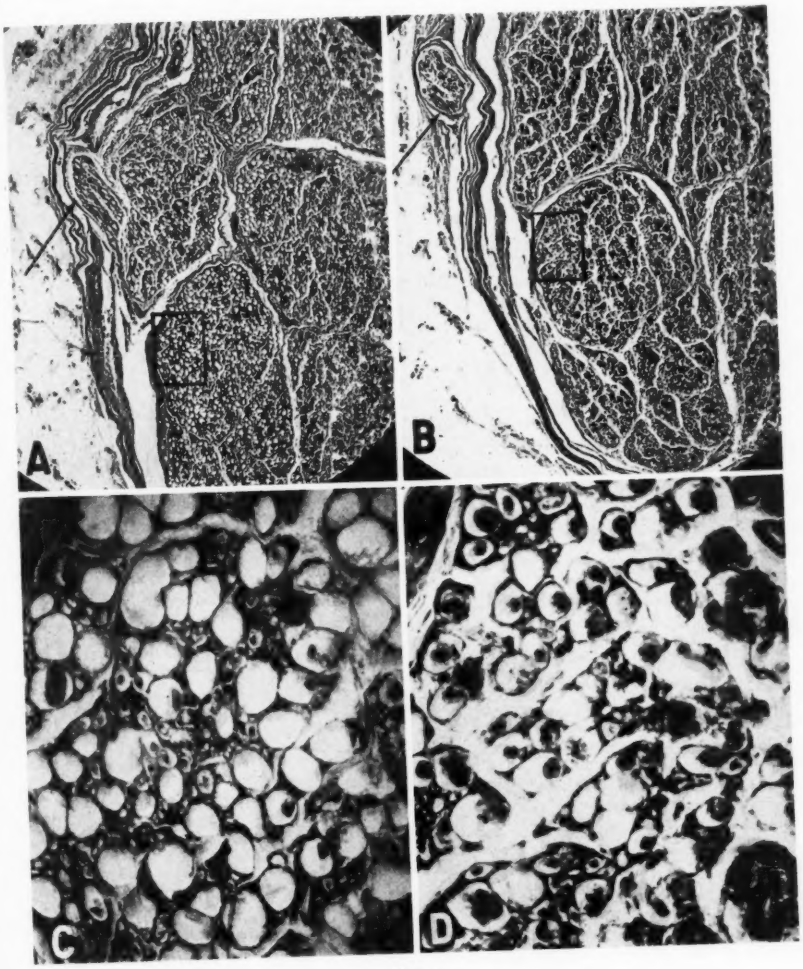

FIG. 1.

in transverse section. In 3E most of the same nerves show evidence of degeneration. Many Schwann tubules are swollen, some are empty and others are filled with multiple rods of Schwann cytoplasm. In one of these tubules 9 distinct Schwann nuclei are visualized (arrow).

The sections in Figs. 3D and E seem to demonstrate, as did those in

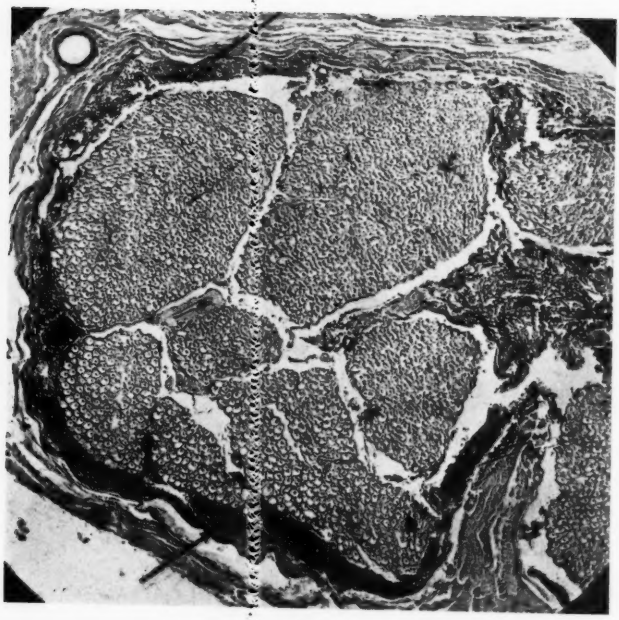

FIG. 2.

FIG. 3A.

FIG. 3B.

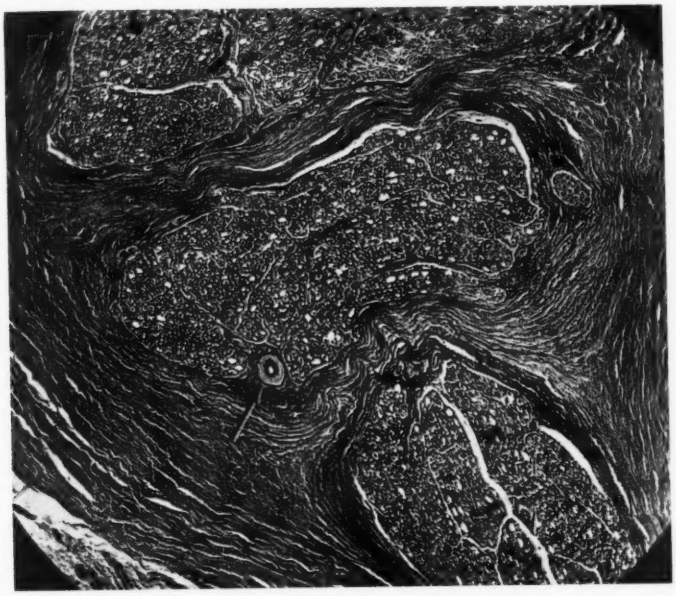

FIG. 3C.

FIG. 3D.

FIG. 3E.

Figs. 1C and D, that apparently a nerve bundle may exhibit different stages of degeneration or normalcy at points only a few mm. apart.

Duncan[1] has shown that gradual constriction of a nerve in young animals destroyed the myelin about the axone but did not completely destroy its function or produce paralysis. However, after $6\frac{1}{2}$ months there was a significant decrease in the size of the limb muscles and a reduction in the number and size of the nerve fibers below the constriction.

Lindblom[5] and Gray[2] have reported both macroscopic and microscopic changes involving the nerve roots removed from constricted foramina. Some of the changes here visualized resemble those described by Holmes and Young[4] in cases of nerve degeneration.

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GLIOMAS INVOLVING THE SPLENIUM OF THE CORPUS CALLOSUM

A ROENTGENOLOGIC STUDY

BENNO SCHLESINGER, M.D.

Neurosurgical Services, Mount Sinai and Bronx Hospitals, New York, New York

(Received for publication November 28, 1949)

ACCORDING to Ostertag,[4] gliomas of the splenium, like other gliomas of the corpus callosum, originate in the region of the superomedial angle of the lateral ventricle. Further growth of the lesion is either systematized or irregular. Systematized growth takes place along the fibers of the corpus callosum, commissura hippocampi and fornix, which may become markedly thickened. Early infiltration of the cingular gyri appears to be the rule. Unsystematized growth may involve the thalamus, the caudate nucleus and the choroid plexus of the lateral ventricles. Sooner or later the lesions may infiltrate most of the posterior portion of both cerebral hemispheres, tumefaction of the splenium being a common property of the entire group.

The roentgenologic features are largely determined by the systematized spread of the neoplasm which, following injection of air into the ventricles or subarachnoid fluid spaces, may permit visualization of characteristically altered parts of their ventricular surfaces.

CASE REPORTS

Case 1. *Bilateral, predominantly left-sided glioma of posterior portion of corpus callosum. Right posterior parietal ventriculography. Partial filling of posterior portion of right vestibule. Block of foramen of Monro.*

Following air injection into the atrium of the right lateral ventricle the lateral views showed a downward displacement of the pineal body, but were otherwise non-contributory, as was the AP view.

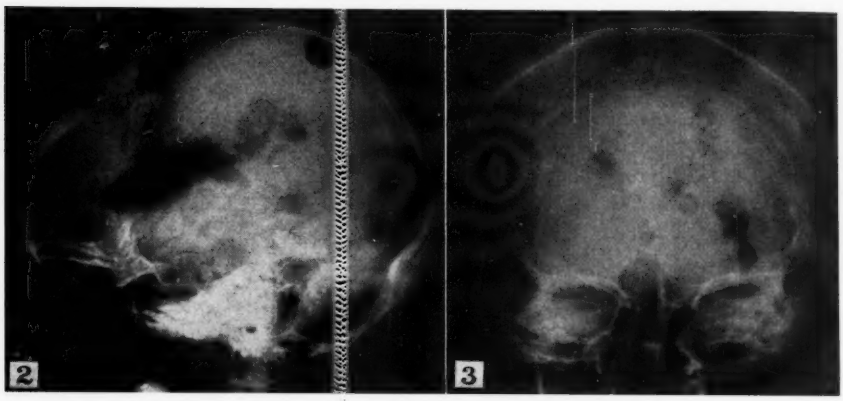
The PA view (Fig. 1) showed the basolateral segment of the posterior portion of the right lateral ventricle only, its superomedial portion being absent. The shadow of the ventricle appeared to be displaced to the right of the median plane, and to be bounded medially by a vertical line. Due to the rotation of the head, which favoured

FIG. 1. Case 1. PA view.

the right side of the skull, the degree of displacement was not fully reflected by this view. For the same reason, the pineal shadow, although displaced to the right, appeared to lie in the median plane. A soft tissue mass, possibly representing the proximal portion of the hippocampus, seemed to protrude into the lower medial portion of the ventricle. On the whole, the image of the ventricle appeared to be enlarged in spite of the absence of its superomedial portion.

At operation, a glioma occupying the depth of the left parietal lobe was found. *Autopsy* revealed spread of the lesion into the opposite hemisphere through the splenium of the corpus callosum.

Comment. The air outlined only that part of the neoplasm that had extended from the left into the right hemisphere but gave no direct informa-



FIGS. 2 and 3. Case 2. Lateral and PA views.

tion as to the location of the lesion in the former. However, the seemingly unsatisfactory ventriculogram confirmed and amplified the clinical diagnosis, as the roentgenologic findings suggested the presence of a tumor of the left occipitoparietal lobe which infiltrated the posterior portion of the corpus callosum and displaced the pineal body downward. Failure of the air to enter the third and the lateral ventricle opposite the site of air injection was attributed to marked swelling of the brain, which narrowed the interventricular foramen.

Case. 2. *Large glioma of splenium involving both parietal and occipital lobes. Downward and forward displacement of pineal body. Unsuccessful ventriculography, followed by lumbar air injection. Filling defect on medial side of vestibule of both lateral ventricles.*

Cannulation of the brain on the left on attempted posterior parietal ventriculography yielded a few cc. of grossly hemorrhagic CSF. Lumbar encephalography was performed a few days later.

The AP views showed a slight symmetrical dilatation of both frontal horns and the ascending portions of the bodies of both lateral ventricles.

Right lateral stereos taken in the recumbent position (Fig. 2) showed, in addition to the enlargement of both anterior horns: (a) sharp depression of the descending portion of the body and the vestibules of the lateral ventricles, more so on the left; (b) slight dilatation of the temporal horns; (c) depression and forward displacement of the pineal gland, which appeared to be projected upon the junction of the temporal horn and the vestibule of the lateral ventricle; and (d) depression of the posterior portion of the roof of the flattened 3rd ventricle. The aqueduct and the floor of the 4th ventricle formed an obtuse angle rather than a curve. A number of elongated shadows converging toward the left vestibule evidently represented needle tracts filled with air.

Lateral views taken in the erect position showed the posterior portion of the 3rd ventricle and the pineal gland to be depressed.

The PA view (Fig. 3) revealed a separation of the posterior portion of the left lateral ventricle by a soft tissue mass, apparently extending into the left vestibule from its medial wall. Above the mass, the previously described composite shadow corresponding to the multiple needle tracts could be seen. The inward and downward slant of the roofs of the lateral ventricles appeared to be increased.

The 4th ventricle appeared to be dilated in both lateral and PA views.

The diagnosis of a bilateral tumor occupying the depth of both parietal lobes was made. It was assumed that the lesions were hemorrhagic and friable, possibly metastatic in character, and that the cannulation of the brain on attempted ventriculography had established a communication between the left lateral ventricle and the interior of the lesion, which became visible following encephalography.

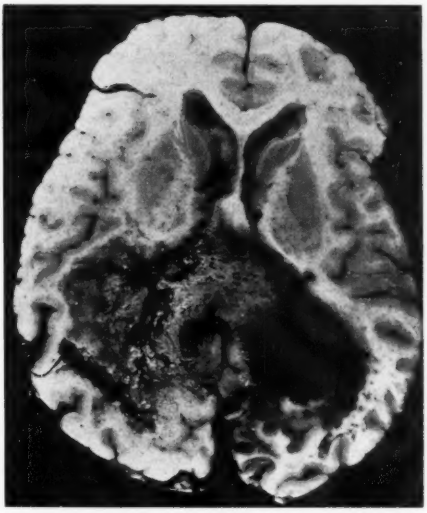


FIG. 4. Case 2.

Autopsy of the Brain. There was a moderate degree of arteriosclerosis of the blood vessels at the base of the brain. Both hemispheres were flabby and the convolutions slightly flattened. Palpation revealed an area of definitely decreased resistance occupying the right upper parietal region.

There was evidence of a mild bilateral pressure cone in both the region of the uncus and the cerebellar tonsils. On sectioning the brain an enormous tumor was found involving the posteromedial portion of both hemispheres (Fig. 4). It extended from about the beginning of the crura of the fornix backward to replace the white matter almost completely, and to undermine the gray matter. The tumor was hemorrhagic and the needle tracts, done on attempted ventriculography, and corresponding to the converging shadows visible on the ventriculograms, could be clearly identified in the disintegrated tumor tissue close to the atrium of the left lateral ventricle. The anterior horns of the lateral ventricles were moderately and symmetrically dilated, their bodies narrow and their posterior horns reduced to slit-like cavities.

Histologic examination proved the lesion to be a typical spongioblastoma multiforme.

Comment. Except for the fact that the posterior portion of the body of the lateral ventricle was invisible in Case 1, the images of the lateral ventricles in both cases were quite similar, because (a) the medial border of the shadow representing the posterior portion of the lateral ventricle was displaced away from the midline; and (b) the normal wing-like form of the ventricular image was converted into a triangle.

Case 3. *Large glioma replacing splenium of corpus callosum, and extending into both occipitoparietal lobes. Frontal ventriculography. Downward displacement of pineal shadow. Widening of shadow of splenium of corpus callosum in transverse diameter.*

AP views revealed a mild symmetrical dilatation of both anterior horns and the adjacent portions of the body of the lateral ventricles. The PA view (Fig. 5) showed

FIG. 5. Case 3. PA view.

FIG. 6. Cramer's Case 6, for comparison with Fig. 5.

the posterior portions of both lateral ventricles, the 3rd ventricle, and part of the body of the left lateral ventricle. A soft tissue mass which occupied the region of the midline could be seen to protrude into both the right and the left vestibule. Due to the superimposition of air present in the body of the left lateral ventricle, the mass could be more clearly visualized on the right. On close inspection some air appeared to be present in the body of the right lateral ventricle, too, but this part of the ventricular system did not contain enough gas to obscure the image of the intraventricular protrusion of the neoplasm.

The 3rd ventricle was in the midline and there was no lateral displacement of the pineal body. The 4th ventricle, as seen in one of the lateral views, was normal.

The pineal gland, as seen in lateral views, was shifted downward and forward, although to a lesser degree than in Case 2.

A preoperative diagnosis of a tumor of the splenium extending into the right parietal lobe was made. At operation, the atrium of the right lateral ventricle was

opened, and the wall of the ventricle found to be pathologically altered. No tumor tissue was removed.

Autopsy of the Brain. The choroid plexuses of the lateral ventricles were displaced laterally by a soft fleshy pinkish-white tumor mass which incorporated the entire splenium of the corpus callosum, more so on the right. The neoplasm extended into the frontoparietal region and as far caudad as the parieto-occipital junction.

On microscopic examination the tumor showed the structure of a highly cellular glioma.

Comment. Since the superomedial portion of the shadows on either side of the midline (representing the posterior portion of the body of the lateral ventricle on either side) were inconspicuous as compared to the shadows of the inferolateral portion, the images of the posterior parts of the lateral ventricles appeared, for all practical purposes, to be represented by the prominent shadows only. The distance between the two vestibules, as seen in PA views, was markedly increased.

DISCUSSION

I. *The Mechanism of Ventricular Distortion in Gliomas of the Splenium of the Corpus Callosum.* As mentioned in the introduction, systematized spread of the lesions under consideration takes place by infiltration of the forceps posterior on either side. The portion of the posterior forceps which sweeps backwards along the medial wall of the atrium and the posterior horn, narrowing its cavity, is known as bulbus cornu posterioris. Studies of coronal sections show that spread of the glioma into the adjacent calcar avis is a very frequent occurrence and that the entire medial wall of both the atrium and the posterior horn is infiltrated. The cavity of the ventricle in the involved region is therefore narrowed (Figs. 4 and 6). If the films are taken in the brow-down position the laterally protruding walls indicating the abnormal separation of the posterior portions of both lateral ventricles are outlined by air. The medial boundary of the narrowed ventricle appears to be altered on account of its vertical course, as in Case 1 or its oblique course, as in Cases 2 and 3. The hydrocephalus of both the anterior and temporal horns is due to narrowing of the foramina of Monro, incident either to edema of the hemispheres, neoplastic infiltration of the fornices or both. If the lesion extends forward toward the midportion of the corpus callosum, the inward and downward slant of the roof of the descending portion of the lateral ventricle appears to be greatly increased, as in Case 2.

II. *Differential Diagnosis.* (1) Pinealomas give rise to a separation of the ventricular shadows similar to that seen in gliomas of the splenium but produce a more marked hydrocephalus, including the front end of the third ventricle, whose caudal extremity is indented by the neoplasm. The roof of the fourth ventricle, if visible, is displaced downward.

(2) Meningiomas of the falx arising in the region of its junction with the tentorium cause depression of the roofs of both lateral ventricles which, however, do not appear to be separated as seen in PA views.

(3) The tip of the superior vermis of the cerebellum lies but a short distance from the lower surface of the splenium of the corpus callosum. Tumors of the superior vermis may produce a pressure cone extending from the infratentorial into the supratentorial space, and a forward shift of the midbrain, which encroaches upon the third ventricle from below and backward. The comma-shaped outline of the third ventricle resulting from this pressure, and the increased distance between the posterior elongated extremity of the third ventricle and the temporal horn are characteristic of this type of lesion. The mechanism of ventricular distortion in tumors of the superior vermis was clarified by Vincent and Le Beau.[6]

(4) Deep lying gliomas of the inferior parietal lobule approaching the splenium cause a marked midline shift, flattening of the entire wing-shaped image of the lateral ventricle as seen in PA views, or indentation of its superolateral wall.[5]

(5) Deep lying gliomas of the occipital lobe approaching the splenium of the corpus callosum produce kinking of the ascending toward the descending portion of the lateral ventricle as seen in lateral views, and marked forward shift on the involved side.

(6) Gliomas of the midportion of the corpus callosum protrude into the ventricle from the junction of the ascending and descending portion of its body.[2] Due to replacement of a large part of the body of the ventricular cavity by a tumor mass, its image may appear to be faint on both AP and PA views, but the separation of the lateral ventricles in PA views is absent.

(7) Poor filling of both lateral ventricles may give the impression of an apparent separation of the images as seen in PA views. However, the size and shape of the posterior horns is quite different from that of the narrowed vestibules, and there are, of course, no changes in the region of the posterior portion of the third ventricle.

CONCLUDING REMARKS

While it appears that the lesions under consideration can be more readily diagnosed by air studies than by clinical methods, the x-ray films do not necessarily tell the whole story. It is true that the location of the lesion is disclosed by its systematized spread as mentioned above, but the irregular spread into caudate nucleus and thalamus may well escape detection. A marked midline shift indicates infiltration by the neoplasm of the opposite hemisphere, as in Case 1. Gliomas of the splenium are no exception to the rule that pathologically altered cerebrospinal fluid spaces made visible by air injection, permit the identification of what may be termed the roentgenologically active part of the lesion but do not preclude the extension of the tumor into adjacent parts of the brain.

The size and extent of gliomas of the splenium, the majority of which are quite vascular, could probably be more accurately determined by bilateral angiography provided both posterior cerebral arteries can be filled. Widening of the arch of the great vein of Galen, or local compression of this

vessel, described by Moniz,[3] may be important diagnostic clues. Displacement of the calcified glomus of the choroid plexus due to encroachment by the enlarged splenium[7] is probably a rare occurrence.

In association with other findings the downward and forward displacement of the pineal body may be a danger signal indicating the presence of vulnerable gliomatous tissue in the posterior portion of the cerebral hemispheres, and should discourage posterior ventriculography by the posterior route.

SUMMARY

1) Roentgenologically, gliomas of the splenium of the corpus callosum appear to be characterized by downward and forward displacement of the shadow of the pineal gland and, following air injection, widening of the distance between the shadows representing the medial walls of the vestibule of the lateral ventricles. The adjacent part of the roof of the lateral ventricles may be sharply depressed on either side of the median plane.

2) The differential diagnosis of the lesions under consideration is discussed.

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GYNECOMASTIA IN PARAPLEGIC MALES

IRVING S. COOPER, M.D.,* COLLIN S. MacCARTY, M.D.,† AND EDWARD H. RYNEARSON, M.D.‡

Mayo Foundation, and Section on Neurologic Surgery, and Division of Medicine, Mayo Clinic, Rochester, Minnesota

(Received for publication December 1, 1949)

GYNECOMASTIA in young adult males rendered paraplegic by trauma to the spinal cord has been reported by Cooper and Hoen,[2] who found an incidence of 22 per cent in a small series of cases. The occurrence of mammary hypertrophy in paraplegic patients has also been noted by Bors,[1] Moehlig[6] and Freeman.[4] The pathogenesis of enlargement of the male breast following injury of the spinal cord has not been elucidated. In order to gain further insight into this syndrome a series of 16 consecutive male paraplegic patients has been investigated. Fifteen of these patients had been rendered paraplegic by trauma to the spinal cord; the remaining patient was completely paraplegic following subtotal spinal cordectomy for an extensive intramedullary glioma.[5] Of these 16 patients, 4 demonstrated bilateral mammary enlargement at some time during the period between time of trauma and time of observation. In 2 additional cases pain and tenderness of the breasts, but no enlargement, developed.

This investigation has been concerned with hormone excretion and other metabolic variations in those paraplegic subjects demonstrating gynecomastia. The 4 cases in which gynecomastia developed are herein briefly reported.

REPORT OF CASES

Case 1. A man, aged 28 years, was rendered paraplegic below the 9th thoracic segment following trauma to the spinal cord incurred in an automobile accident. Bilateral enlargement of the breasts appeared 11 months after injury and gradually lessened during the ensuing 4 months. Hormone determinations during the period when the gynecomastia was regressing revealed the urinary excretion of the 17-ketosteroids to be reduced to 2.2 mg. in 24 hours (normal value 6 to 20.8 mg.) and the urinary corticosteroid excretion to be 0.30 mg. in 24 hours (normal value 0.21 to 1.16 mg.). Neither prolan nor estrogens could be demonstrated in the urine. The BMR was −21 per cent and the serum protein was 6.0 gm. per 100 cc. with an albumin-globulin ratio of 1:1. The testicles measured 4 by 2 cm. (normal measurement 4 to 5 cm. by 2.5 to 3 cm.).

Case 2. A man, aged 44 years, experienced an injury of the spinal cord and consequent paraplegia below the 6th thoracic segment when a vehicle in which he was riding overturned on Sept. 1, 1948. In January, 1949, bilaterally enlarged painful, tender breasts developed. The mammary enlargement and discomfort regressed

* Fellow in Neurosurgery, Mayo Foundation.
† Section on Neurologic Surgery, Mayo Clinic.
‡ Division of Medicine, Mayo Clinic.

in 1 month. The urinary excretion of 17-ketosteroids was 1.5 mg. in 24 hours; that of corticosteroids was 0.40 mg. Prolan could not be demonstrated in the urine. There were 16 rat units of urinary estrogens in 24 hours (normal value 5 to 25 rat units daily). The serum protein was persistently recorded between 4.5 and 5.1 gm. per cent with an albumin-globulin ratio of 0.6:1 to 1.0:1. The testicles measured 3.5 by 2 cm.

Case 3. A male, aged 17 years, experienced a transection of the spinal cord at the 5th cervical segment when he struck his head while diving into shallow water. Twelve months after injury painless enlargement of both breasts developed but this disappeared in 3 to 4 weeks. Daily urinary excretion of 17-ketosteroids was 5.0 mg. while that of corticosteroids was 0.40 mg. Neither prolan nor estrogens could be demonstrated in the urine. Serum protein concentration was 6.0 gm. per 100 cc. with an albumin-globulin ratio of 1:1. The testicles measured 3.5 by 2.5 cm.

Case 4. A man, aged 27 years, underwent thoracic, lumbar and sacral spinal cordectomy because of the cephalad progression of an intramedullary glioma. Three months after the surgical procedure bilateral gynecomastia was noted (Fig. 1). Hormone determination revealed the excretion of urinary 17-ketosteroids to be 1.9 mg. in 24 hours and that of the corticosteroids to be 0.22 mg. Neither prolan nor estrogens could be recovered from the urine. The serum protein totaled 5.8 gm. with an albumin-globulin ratio of 1:1. The BMR was −22 per cent. The testicles measured 4 by 2.5 cm. This patient died from multiple intracranial gliomas. Postmortem examination revealed gynecomastia and atrophy of the testicular tubules with aspermatogenesis as well as atrophy of the pituitary gland, adrenal cortices and prostate. The metabolic variations in this patient following spinal cordectomy are reported in detail elsewhere.[3]

FIG. 1. Mammary enlargement present in a man, aged 27 years, 3 months after subtotal spinal cordectomy.

COMMENT

The incidence of gynecomastia in 16 consecutive cases of hospitalized paraplegic patients was 25 per cent. Examination of these patients revealed each to have abnormally low urinary excretion of the 17-ketosteroids with the excretion of corticosteroids in the low normal range. Hypoproteinemia with reversal of the albumin-globulin ratio was present in each case. The basal metabolic rate was obtained for 2 of the 4 patients and was −21 and −22 per cent in these patients. Testicular atrophy was grossly or microscopically evident in each case. The significant findings are summarized in Table 1.

Speculation regarding the pathogenesis of gynecomastia in traumatic paraplegic patients has been considered elsewhere[2,3] and will not be repeated here. Decreased excretion of the 17-ketosteroids, hypoproteinemia and lowered basal metabolic rate have been observed in traumatic paraplegic patients without gynecomastia, and the degree of abnormal variation of these

TABLE 1

Significant metabolic data for 4 male paraplegic patients with gynecomastia

	Case			
	1	2	3	4
Time of appearance of gynecomastia, months after injury	11	5	12	3
Urinary 17-ketosteroids, mg. in 24 hours (normal value 6–20.8 mg.)	2.2	1.5	5.0	1.9
Urinary corticosteroids, mg. in 24 hours (normal value 0.21–1.16 mg.)	0.39	0.40	0.40	0.22
Basal metabolic rate, per cent	−21	Not obtained	Not obtained	−22
Urinary prolan	Negative for normal amounts	Negative for normal amounts	Negative for normal amounts	Negative for normal amounts
Urinary estrogens	Negative for normal amounts	16 rat units in 24 hours	Negative for normal amounts	Negative for normal amounts
Testicular atrophy	Grossly evident	Grossly evident	Grossly evident	Microscopically evident
Serum protein level, gm. per 100 cc.	6.0	4.5–5.1	6.0	5.8
Albumin-globulin ratio	1.0:1	0.6–1.0:1	1.0:1	1.0:1

findings usually appears directly proportional to the severity of injury of the spinal cord.

SUMMARY

Gynecomastia has been observed in 4 young men after severe trauma to the spinal cord. In each instance there was decreased urinary excretion of the 17-ketosteroids and hypoproteinemia. Each of these men presented some degree of testicular atrophy. The basal metabolic rate was obtained in 2 of these cases and was significantly decreased in each instance. The observed gynecomastia, decreased excretion of the 17-ketosteroids, lowered basal metabolic rate, testicular atrophy and hypoproteinemia are considered to be sequelae of injury to the spinal cord.

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A CONTRIBUTION TO THE PHYSIO-PATHOLOGY OF THE CHOROID PLEXUS*

LOYAL DAVIS, M.D.

Department of Surgery, Northwestern University Medical School, Chicago, Illinois

(Received for publication December 7, 1949)

And though the information is not likely to be forthcoming, at least from clinical cases of icterus, I believe that even when a trace of bilirubin can be detected in the subarachnoid fluid it will not be found in the ventricles; and if this should prove to be so, it would indicate that the pigment finds its way into the arachnoidea by way of the perivascular spaces rather than by passage through the filter of the plexus.

HARVEY CUSHING

IN THE first of three Cameron Prize Lectures delivered in 1925 at the University of Edinburgh, Harvey Cushing[1] discussed the formation of biliary pigments with relation to the cerebrospinal fluid.

Mestrezat[2] called attention to the presence of bile pigments in the spinal fluid of patients with deep jaundice of long standing, and this has become an accepted fact. As he pointed out, the level of the blood icterus in relation to the appearance of bilibrubin in the spinal fluid varies considerably with the individual patient. Normally, the bile pigments do not pass the barrier of the choroid plexus. Mestrezat assumed that when the spinal fluid was discolored in deeply jaundiced patients there was damage of the choroidal epithelium which made it hyporesistant to the passage of the bile pigments, even though the damage could not be demonstrated by histopathological methods.

It has not been known, however, whether or not the ventricular fluid contains bile pigments in chronically jaundiced patients. As Cushing proposed the question, the answer would indicate that he believed that the pigment finds its way into the arachnoidea by way of the perivascular plexus and, therefore, that the choroid plexus would remain a constant barrier.

Attempts to answer this question experimentally and by the examination of the ventricular fluid of deeply jaundiced patients have been made since 1926. It was not difficult to produce experimentally a jaundiced cat, dog or monkey by obstructing the common bile duct; the urine, blood serum, and ocular fluid showed the presence of bile pigments. In each instance in which the blood bilirubin was high the spinal fluid contained bilirubin. The difficulty of performing an accurate and meticulous ventricular puncture in these animals to obtain a quantity of fluid sufficient to make an accurate determination of bilirubin made it obvious that the ventricular fluid of only human patients would provide a satisfactory answer.

The bilirubin content of the ventricular and spinal fluids and of the blood serum was determined by the indirect van den Bergh method in 3 patients.

* Aided by the Grunow Surgical Fund.

In all instances a direct van den Bergh test was also made on the blood serum. Two of the patients had cirrhosis of the liver and the third patient had an obstructive jaundice due to a carcinoma of the pancreas. Bilirubin was found to be present in the ventricular and spinal fluids of each of the patients in milligram-percentages comparable to the blood serum content, as is shown in Table 1.

TABLE 1

	Spinal Fluid Bilirubin	Ventricular Fluid Bilirubin	Blood Bilirubin van den Bergh		
	Indirect van den Bergh		Direct 1 min.—30 min.		Indirect
	mg. %	mg. %	mg. %	mg. %	mg. %
Patient, A.B. Liver cirrhosis (verified)	.06	.007	4.6	6.2	7.34
Patient, M.B. Liver cirrhosis (verified)	.02	.008	3.8	5.78	10.9
Patient, L.C. Common duct obstruction Carcinoma of pancreas (verified)	.03	.028	16	20.6	26

The bilirubin content of the spinal fluids showed no direct relationship to the level of the blood serum bilirubin, but such a direct variation might be indicated between the blood serum and the ventricular fluid. In each instance the spinal fluid bilirubin was higher than that in the ventricular fluid and this might be explained by the more active circulation of the ventricular fluid and the addition of bilirubin to the spinal fluid from the perivascular spaces.

The evidence that bilirubin does pass the barrier of the choroid plexus from the blood into the ventricular fluid in patients with jaundice of long standing and that this passage is made possible by functional damage of the choroidal epithelium appears to be conclusive and in keeping with knowledge concerning the physiological functions of the choroid plexus.

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CASE REPORTS

FIBROCASEOUS CEREBELLAR TUBERCULOMA

SURGICAL RESECTION IN CONJUNCTION WITH STREPTOMYCIN THERAPY*

DONATHAN IVEY, M.D., SAMUEL PHILLIPS, M.D., AND ARNOLD M. MEIROWSKY, M.D.
Section on Neurological Surgery, Surgical Service, and Section on Tuberculosis Medical Service, Veterans Administration Medical Teaching Group, Kennedy Hospital, Memphis, Tennessee

(Received for publication October 20, 1949)

Ever since Hahn[14] in 1880 made the first attempt to extirpate a cerebral tuberculoma, there has been much discussion as to the applicability of surgical measures to tuberculous lesions of the brain. While most authors agree that a circumscribed tuberculoma near the cerebral surface lends itself well to total resection,[2,5,9] Van Wagenen in his classical review questions the value of extirpation as a curative measure.[12] The latter, as well as Buchstein and Adson,[2] particularly stresses the discouraging results from surgical resection of the cerebellar tuberculoma. Recurrence of symptoms or postoperative tuberculous meningitis resulting in death has been the usual sequel to partial or attempted total resection of a cerebellar lesion.[2,8,12] Buchstein and Adson[2] list technical difficulties and structural peculiarity of cerebellar tuberculomas as reasons for poor results. An exception in the literature is Frazier's case,[11] the patient having been reported by Kwan[6] as living and well 11 years after total extirpation of a large, solid cerebellar tuberculoma.

Sufficient evidence has been accumulated to show that streptomycin may be an effective therapeutic agent in the treatment of tuberculous meningitis.[3,7] It has also been shown that within 90 days the tubercle bacillus tends to develop strains resistant to streptomycin in the majority of patients.[4] Streptomycin does not readily penetrate into brain substance.[1] On the basis of experimental evidence, Rhymer, Wallace, and co-workers[10,13] have proposed the actual presence of a streptomycin inhibitor within the cerebral tissue. One may therefore presume that streptomycin might prevent or cure a postoperative tuberculous meningitis, but that it will not be effective in the treatment of an existing intracerebral tuberculoma. Obrador and Urquiza,[8] in a recent report of a tuberculous abscess, have already stressed their impression that streptomycin may become a powerful ally of surgical technique in the treatment of tuberculomas of the brain and that it may ward off the bacillar spread in the cerebrospinal fluid which so frequently follows attempts at removal.

It seems, then, that the advent of streptomycin throws a new light on the applicability of surgical measures to tuberculous lesions of the brain. Little can be expected from any partial resection of an intracranial tuberculoma. Total surgical extirpation, however, in conjunction with intramuscular and intrathecal streptomycin therapy as applied in the case to be presented, might well prove to enhance the life expectancy of patients with tuberculous lesions of the brain.

* Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are a result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

CASE REPORT

A.T., a 25-year-old, right-handed, colored veteran, was admitted to Kennedy Veterans Administration Hospital on March 19, 1948, with a history of intermittent, throbbing, bifrontal headaches, which first occurred in 1945. In January 1948, they increased in severity. The patient began to stagger and to walk on a broad base. Two weeks prior to admission he started to vomit and to complain of blurred vision. Members of the family had noticed some slurring of his speech. Twenty-four hours prior to admission double vision occurred on looking to the right.

Examination. The patient was rather poorly developed and undernourished. He was rational and oriented, but appeared drowsy. His speech was thick and slurred. Scalp and skull were tender to pressure and percussion. There was bilateral papilledema of 2–3 D. The 6th nerve was paralyzed on the right and slightly impaired on the left. Flattening of the nasolabial fold was suggestive of an upper motor neuron paresis of the left 7th nerve. The 11th and 12th nerves were partially paralyzed on the right. There was considerable dysmetria of right upper and lower extremity. Barré's sign was positive on the right. Romberg was positive. The patient walked on a broad base, staggering and falling to either side. There was diminution of two-point discrimination and position sense in the right lower extremity. Deep tendon and superficial reflexes were equally active bilaterally; there were no pathological toe signs and no clonus. The patient had moderately severe photophobia, but showed no other meningeal signs.

The systemic findings were normal. It is noteworthy that the chest film did not show any pulmonary disease. The provisional diagnosis of a space-occupying lesion involving the right cerebellar hemisphere seemed well established.

On March 22, 1948, bilateral posterior burr holes were made. Ventriculography on March 23 showed symmetrical dilatation of the lateral ventricles, dilatation of the 3rd ventricle, and obstruction of the ventricular system at the midportion of the aqueduct of Sylvius. The patient tolerated the procedure poorly and showed signs of decompensation. It was necessary to postpone surgical treatment and to allow the patient to compensate for the severely increased intracranial pressure by continuous ventricular drainage. Within 3 days, blood pressure, pulse, and respiration became stable.

1st Operation. On March 30, 1948 a bilateral suboccipital craniectomy was performed (A.M.M.). The dura was opened over the right cerebellar hemisphere and found to be loosely attached to the arachnoid, which was quite firmly adherent to an underlying yellow-grayish granulomatous mass. Frozen sections identified this as a fibrocaseous tuberculoma. The operation was limited to decompression and biopsy.

Postoperative Course. The patient was given streptomycin, 0.4 gm. intramuscularly 5 times daily, and 50 mg. intrathecally 3 times per week. He also received 4 gm. of promin intravenously daily with 1 week's rest interval between each 2 weeks of promin administration. On the strength of previously cited experimental and clinical evidence as to the effectiveness of streptomycin in the prevention of tuberculous meningitis, [1,3,4,7,10,13] the optimum date for total resection of the granuloma was arbitrarily set 30 days after the initiation of the streptomycin treatment.

2nd Operation. On April 30, 1948 a second-stage suboccipital craniectomy was performed (A.M.M.). Except for a narrow space, containing clear yellow fluid over the right cerebellar hemisphere, dura and galea were attached and the flap had to be dissected away from the dura. The dura overlying the right cerebellar hemisphere was covered with an organized yellowish exudate. In a diameter of 3 cm., covering the lateral and central aspect of the hemisphere, the dura was firmly adherent to the underlying tuberculoma. After the dura had been resected, the granuloma was found to extend rostrally to the tentorium and laterally to the meninges. Medially and rostrally, atrophic and grayish discolored cerebellar cortex could be recognized. The only part of the right cerebellar hemisphere that did not seem to be involved by the granuloma was found caudally and medially. In an attempt at total resection of the granuloma, four-fifths of the hemisphere had to be removed, leaving a narrow strip of cere-

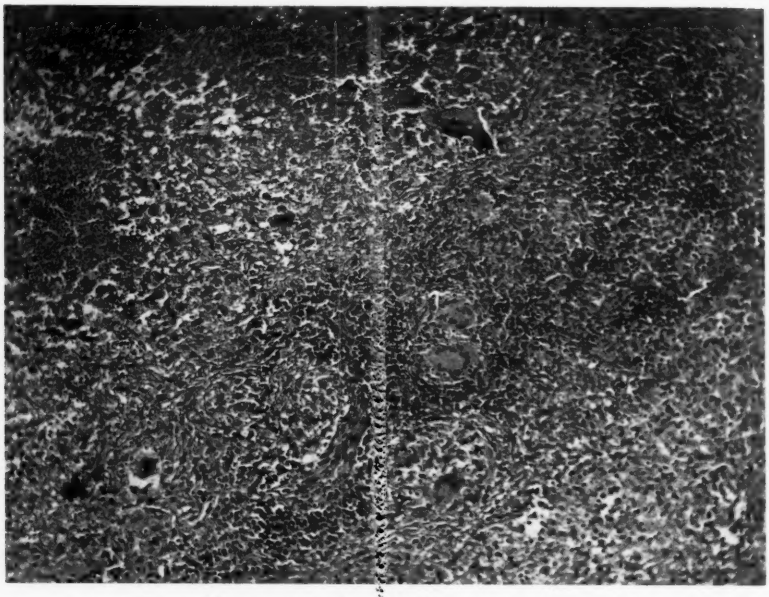

FIG. 1. Microscopic section of the fibrocaseous tuberculoma.

bellum medially and caudally. The cut surface of this area was cauterized. Preserved dura was used for closure of the defect. The patient withstood the operation well.

Histological Studies (Fig. 1). There were extensive areas of caseation necrosis surrounded by numerous epithelioid cells, fibroblasts, Langhans' giant cells, and chronic inflammatory cells. In the surrounding brain tissue, marked gliosis could be seen as well as perivascular round-cell infiltration. A few compound granular corpuscles were noted. Acid-fast stain revealed the presence of tubercle bacilli (Fig. 2).

FIG. 2. Acid-fast stain showing (arrows) tubercle bacillus within tuberculoma.

Course. The administration of streptomycin intramuscularly and intrathecally, and of promin intravenously, was continued postoperatively until a 120-day period of treatment had been completed. The patient made an uneventful recovery. He gained weight steadily. Except for a persistently slurred speech, all neurological signs disappeared. He was discharged on Aug. 12, 1948.

On Sept. 15, 1948 the patient returned to Kennedy Hospital, complaining of pain in the lumbar region. Neurological examination was entirely negative. There was no evidence of recurrence of the intracranial tuberculoma. He had marked localized tenderness over the 4th lumbar vertebra, moderate spasm of the paravertebral lumbar muscles, and severe pain on straight leg raising. X-rays revealed a moth-

eaten appearance of the 4th lumbar vertebra which seemed typical of a localized osseous tuberculosis. He was transferred to the orthopedic service and placed in a cast. In December 1948, a spinal fusion was performed. The patient has remained afebrile and asymptomatic. At the time of writing, 17 months after total resection of the cerebellar tuberculoma, there has been no evidence of recurrent tuberculous disease of the central nervous system.

SUMMARY AND CONCLUSIONS

1. The pertinent literature with regard to the surgical treatment of intracranial tuberculomas and the effectiveness of streptomycin in combating tuberculous lesions of the central nervous system has been reviewed.

2. The case history of a patient with a fibrocaseous tuberculoma involving the greater part of the right cerebellar hemisphere has been reported. The applied treatment, consisting of total resection of the tuberculoma in conjunction with streptomycin therapy, has been discussed.

3. It is suggested that total resection of an intracranial tuberculoma in conjunction with streptomycin therapy may alter the poor prognosis of tuberculous lesions of the central nervous system.

The pathological sections were studied and diagnosed by Dr. D. James Greiner.

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OLIGODENDROGLIOMA IN PRE-ADOLESCENCE

REPORT OF A CASE

HARVEY GASS, M.D., AND WILLIAM P. VAN WAGENEN, M.D.
Department of Surgery, Neurosurgical Division, Strong Memorial Hospital, and The University of Rochester School of Medicine and Dentistry, Rochester, New York

(Received for publication November 10, 1949)

The occurrence of an oligodendroglioma in childhood is unusual. We have been able to find in the literature only 7 cases of this type of tumor in pre-adolescents.

Dickson[4] reported a 6-year-old boy with such a tumor in the floor of the third ventricle; the symptoms were of 4 months' duration. Bailey and Cushing[2] mention a 5-year-old child with an oligodendroglioma of the brain who had had symptoms for 17 months. Bailey and Bucy[1] refer to 1 possible example, also in a child 5 years old. Love[6] reported a tumor occurring in a 13-year-old boy who, however, had had petit mal seizures from the age of 5. Beck and Russell[3] collected 2 cases of oligodendrogliomatosis, with diffuse spread of the tumor over brain and spinal cord, occurring in 2 girls, one aged 6½ whose duration of illness was 10 weeks, and the other, aged 4, who had been sick for 6 or 7 months when first seen. Finally, Hughes and Couper[5] reported a tumor in an 8-month-old male infant, who had been known to have a squint for 2 weeks. This tumor, however, in portions appeared to be a medulloblastoma as well as an oligodendroglioma. It occurred in the midbrain.

CASE REPORT

S.M.H. #291439. E.D., an 8-year-old right-handed boy, was admitted to the Strong Memorial Hospital on Feb. 1, 1949 because of epilepsy. He was apparently well until the age of 15 months when it was noted that he had several petit mal spells daily. About 4 months prior to admission, he also started having grand mal seizures occurring about once weekly. Both types of attacks were often precipitated by excitement and were sometimes ushered in by a warning of epigastric pain.

At the age of 15 months he was able to say a few words but did not enlarge his vocabulary until the age of 5. Bowel and bladder training were not accomplished until then. He never developed sufficiently to go to school, and engaged in but a few games. He played mostly by himself. He was able to feed, wash and dress himself. He was considered to be "nervous and touchy" by his parents.

Anticonvulsant therapy (phenobarbital) was started 4 months before admission but was of little help in controlling his seizures.

There was a strong family history of epilepsy and nervous disorder. His maternal grandmother, great grandmother, aunt and uncle and a paternal cousin had all suffered from epilepsy. The mother at the age of 3 and a maternal uncle were said to have had "nerve spells" of some kind. The patient's 3 siblings were living and well.

Examination. He was well developed and nourished, but was unable to understand or carry out simple commands unless they were demonstrated. He used only single words or short phrases and spoke these with defective, sometimes unintelligible pronunciation. His speech was frequently profane and often inappropriate. The entire examination was otherwise within normal limits.

Laboratory Studies. Blood studies, serum calcium, phosphorus values and urinalysis were all normal.

CSF was under a pressure of 158 mm. of water and showed 2 WBC/c.mm. and 20 mg. per cent protein.

Roentgenograms of the skull (Fig. 1) revealed numerous irregular calcifications lying adjacent to the floor of the right anterior fossa. The superior orbital margin was less pronounced on the right than on the left side.

Operation. On Feb. 9, 1949, under endotracheal ether anesthesia, a right frontoparietal bone flap crossing the midline was turned down. The dura was opened widely up to the superior sagittal sinus. The right frontal lobe was removed down to the ventricle. The tumor was found to be participating in the formation of the anterior horn of the ventricle, particularly on the lateral side, and seemed to be invading the head of the caudate nucleus. It was removed in piecemeal fashion, protecting the basal ganglia as much as possible. At the end of the dissection the great wing of the sphenoid was in the field. The right optic nerve had been cut off near the trigone and the various branches of the anterior cerebral and of the middle cerebral artery going to the frontal lobe had been actually ligated. The wound was closed in the usual fashion after covering the exposed brain with Gelfoam film.

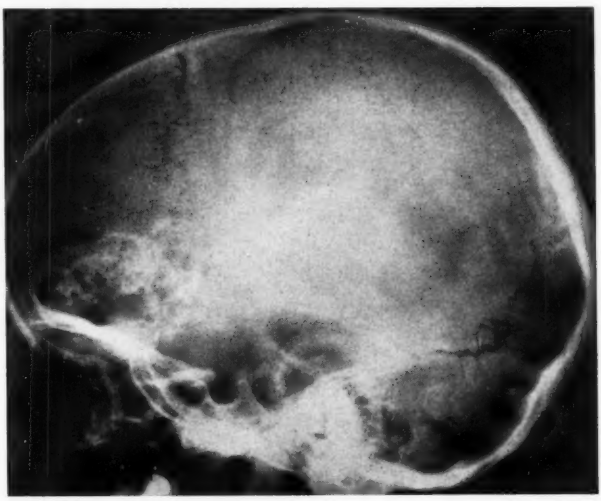

FIG. 1. Calcification of an oligodendroglioma in the right frontal lobe of an 8-year-old boy.

Postoperative Course. There was rather persistent nuchal rigidity and moderate febrile reaction, requiring penicillin and frequent tapping of the flap for about 10 days.

He was discharged on dilantin 0.1 gm. 2/day and phenobarbital 0.032 gm. 3/day 21 days after operation. At that time his neck was supple, the wound was well healed and there was no fluid beneath the flap. Gross neurological findings were negative except for his mental state. He was ambulatory and fed himself. He had not yet re-learned to call for the bed pan so that he frequently wet or soiled the bed. His speech was unchanged from his preoperative status. He was alert and active and played quietly in bed by himself. He followed simple commands rather readily and was generally cooperative. He had had no seizures.

When seen 2 months postoperatively he still had had no seizures. His mother reported a remarkable improvement in the boy's behavior and this was apparent to us also. He had gained 6 pounds in weight and he was pleasant, quiet and agreeable. He evidently understood and followed simple directions, and if the conversation was in simple words, he could give prompt appropriate simple answers. His mother stated that he no longer preferred playing by himself and that he had ceased teasing his younger sister. He obeyed directions at home, no longer used profanity, and in all respects except use and comprehension of a normal vo-

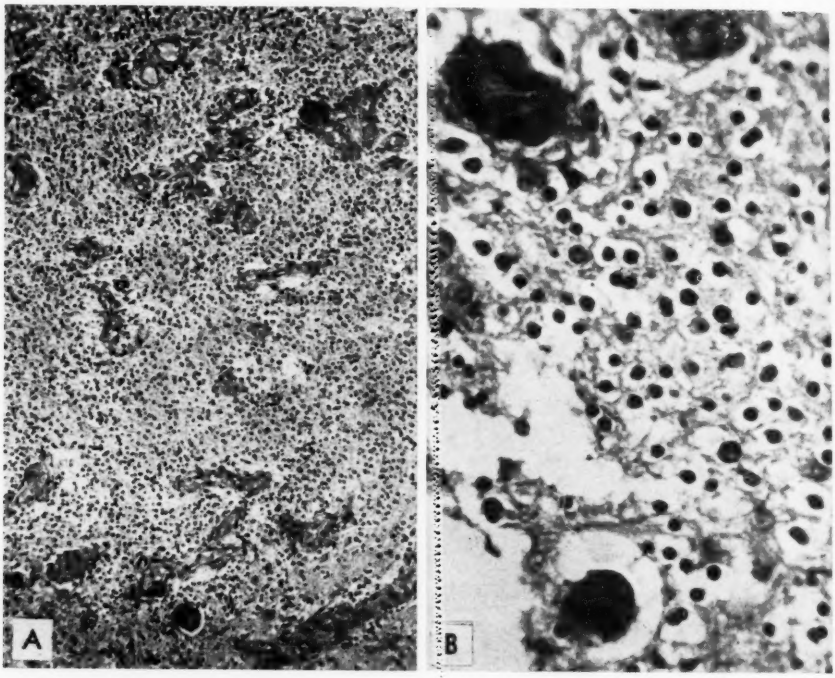

FIG. 2. Photomicrographs of tumor. (A) Showing sheets of closely set oligodendroglial cells. A small calcium deposit is in lower part of field ×110. (B) Higher magnification showing typical round nuclei, perinuclear halos, and deposits of calcium. ×430.

cabulary for his age, he appeared normal. It was felt that even this deficiency could now be amended with proper education.

Pathological Diagnosis: Oligodendroglioma (Fig. 2).

SUMMARY

A case report is presented of an extensively calcified oligodendroglioma occurring in the right frontal lobe of an 8-year-old boy. The onset of symptoms occurred at the age of 15 months. It is assumed that the onset of tumor growth was near the time of the first convulsion. Seven other cases of oligodendroglioma occurring in pre-adolescent children have been found in the literature.

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METASTATIC PINEALOMA INVOLVING THE OPTIC CHIASM

GEORGE S. BAKER, M.D., AND C. WILBUR RUCKER, M.D.
Section on Neurologic Surgery, and Section on Ophthalmology, Mayo Clinic, Rochester, Minnesota

(Received for publication November 14, 1949)

When blindness results from a brain tumor, recovery of vision seldom ensues, even after removal of the tumor. An exception occurred in a case in which a pinealoma had metastasized to the optic chiasm and optic nerves. The bizarre manner in which pinealoma may affect the central nervous system has been stressed by Stringer,[5] Baggenstoss and Love,[1] Kubik,[3] Russell[4] and Troland and Brown.[6] Horrax and Wyatt[2] have described ectopic masses of pinealomas about the optic chiasm.

REPORT OF A CASE

A boy 17 years old complained of rapid loss of vision during a period of 3 weeks. For 1 week he had suffered headache of moderate intensity. Polydipsia and polyuria had been present 2 years, and had been controlled by the patient's local physician with pitressin tannate and pituitrin.

On examination at the Mayo Clinic the right eye was found to be blind, and the acuity of the left eye was recorded as 2/60. The field of the left eye was restricted to a small nasal isle (Fig. 1). The optic disks appeared to be normal. Roentgenography of the skull showed no evidence of disease. Neurologic examination disclosed no abnormalities. The tentative diagnosis of craniopharyngioma or chiasmal arachnoiditis was suggested, and surgical exploration was advised. During the course of these examinations the patient's vision continued to fail, and for 24 hours prior to operation he was completely blind and his pupils did not react to light.

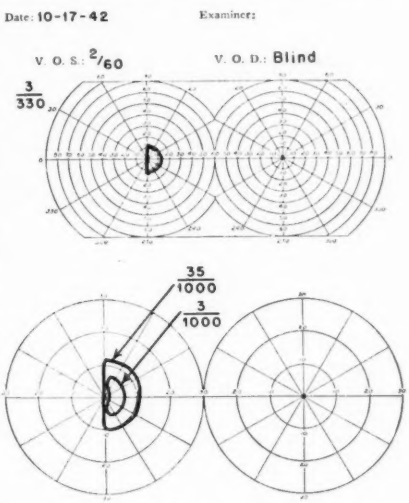

FIG. 1. Field of vision on first examination 3 days prior to operation.

Right transfrontal craniotomy was performed and a tumor mass was found completely surrounding both optic nerves and the chiasm. The growth was friable, bright red, and was readily removed from the area by suction and gentle teasing with pituitary forceps. The pathologic report was "pinealoma."

Recovery of vision during the patient's postoperative period was remarkable. Within 2 days he could see light, and by the 3rd day could distinguish moving objects. By the 5th day he could read large letters with both eyes. On the 16th day visual acuity was recorded as 6/7 with the right eye and 6/20 with the left eye. The fields of vision showed only a moderate contraction (Fig. 2).

A course of roentgen-ray therapy was administered to the skull as is done in the presence of primary pinealomas. Six weeks after operation a second course of roentgen-ray therapy was administered, the fields of vision were again charted and were found at that time to be normal (Fig. 3). One month later the visual acuity was still normal and roentgenography of the skull showed evidence of calcification and enlargement of the pineal gland.

One year after surgical operation the patient complained of pain in the back and aching in his legs. This was regarded as a result of a metastatic lesion in the spinal cord. Deep roentgen-ray therapy was administered to the entire spinal column. The patient's vision was still normal. For about a month there was slight abatement in symptoms referable to the spinal cord, and then paraplegia gradually developed in spite of roentgen-ray therapy. The patient died within the next year.

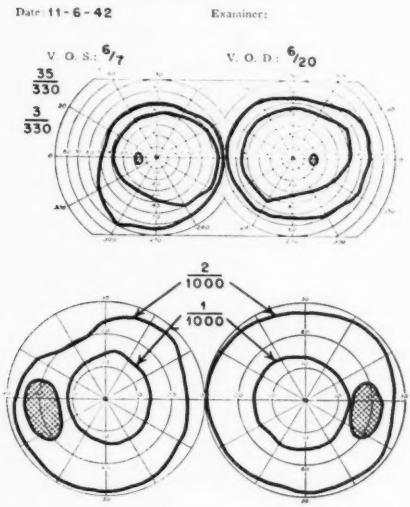

FIG. 2. Field of vision 16 days after operation.

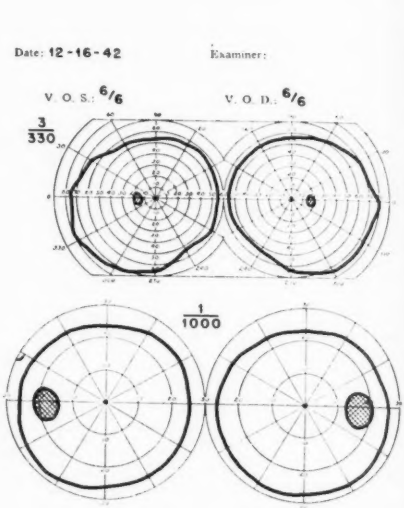

FIG. 3. Field of vision 6 weeks after operation.

CONCLUSION

This case represents some of the usual features of the course of pinealoma, but is unusual in that it furnishes an example of (1) recovery of vision by means of surgery after complete blindness, and (2) maintenance of good vision during the 2 years the patient survived.

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BOOK REVIEW

DIAGNOSIS AND TREATMENT OF BRAIN TUMORS AND CARE OF THE NEUROSURGICAL PATIENT. By Ernest Sachs. *St. Louis: C. V. Mosby Co.*, 1949, 2d ed., 552 pp., 348 figs., and 10 color plates. Price, $15.00.

This volume on intracranial tumors, now in its second edition, was written primarily for the medical student, house officer, and the medical profession in general. It presents an honest picture of what can and cannot be accomplished surgically. Inclusion of detailed case reports of successfully as well as unsuccessfully treated patients keeps the presentation on a realistic level.

This second edition contains essentially the same material as the first edition (1931) with minor alterations. Additions include concise descriptions of pneumoencephalography, electroencephalography, angiography, intracranial aneurysms and intraventricular tumors. The originally brief section on instruments, surgical technique, and postoperative care has been greatly expanded, and twenty-three well documented case reports have been added. Finally, this edition contains two new chapters which briefly consider miscellaneous neurosurgical subjects including spinal cord tumor, subdural hematoma, brain abscess, trigeminal neuralgia, hydrocephalus, compound fractures of the skull, cranioplasty, Jacksonian epilepsy, ruptured intervertebral disc, chordotomy, spina bifida, peripheral nerve repair, spinal cord injury and rhizotomy for spastic muscles.

The outstanding feature of the volume is the style in which it is written. It is blunt, dogmatic and rambling. Like most forceful teachers Sachs presents in a firm manner his own viewpoint even though it may be one not generally held. For example,

p. 482. All laminectomy operations are best carried out with a patient lying on his abdomen.

p. 333. I do not believe in giving any preliminary drugs when doing an operation under local anesthesia.

p. 477. Nevertheless, they [tantalum plates] are not as satisfactory as autotransplants [outer table of skull and attached periosteum], and I have never yet seen a defect so large that it could not be covered by an autotransplant.

p. 340. . . . Avertin combined with Novocain constitutes an ideal anesthetic for all neurosurgical cases . . . .

p. 527. . . . one-eighth of a grain [of morphine] . . . is too small a dose to affect the respiratory center.

At times lavish in his praise of the viewpoint of a contemporary, Sachs at other times may be bluntly critical of the opinion of the same person. Interesting examples of the latter are:

p. 535. He [McKenzie] believes in closing the dura completely after a partial removal [of glioblastoma], with the idea that when the tumor recurs, the patient will die more quickly. My objection to this procedure is that, when the patient's tumor recurs, if the dura has not been left open, he will suffer just as much as before he was operated upon, and frequently will lose his eyesight.

p. 536. He [Cushing] has recorded cases in which he has operated repeatedly; in one instance sixteen times over a period of years. This, I think, is going too far . . . if I cannot remove a glioma completely on a second attempt, I rarely try again.

p. 461. I have had opportunity to examine a number of his [Dandy's] patients and, in every instance, have found [contrary to his claims] an area of anesthesia similar to that found after section of the root in the middle fossa.

p. 464. Adson ligates it [middle meningeal artery], but I have found it simpler to put on a silver clip and then coagulate it.

Every statement in the book is made with such good intent and sincerity that it is unlikely that those criticized would be offended even by the following:

p. 526. It has been a repeated source of amazement to me how ignorant young house officers are in taking care of bedsores. It is a serious criticism of the methods of instruction in vogue in our medical schools.

p. 524. . . . today most supervisors of nurses, and nurses themselves, alas! do not feel as keenly as they used to that bedsores are a reflection on the nursing.

p. 513. It [application of good binders] is almost a lost art, like bandaging among house officers . . . .

Although written in a strictly serious vein, the book contains numerous comments that will cause former students of Sachs to smile in recollection of some of their experiences with this forceful and colorful teacher. For example (p. 337) " . . . a cranial operation, under local anesthesia, is a most trying experience. It is a great ordeal for the patient, and very hard on the surgeon, particularly if he is somewhat high-strung," or (p. 521) "In spite of a very conscientious group of house officers, there always are cases every year in which I have found a rectal impaction."

Sachs, who retired from active surgical practice in 1949, has based his comments on the experiences of a long and distinguished career which began when his specialty was young. He has done well to write so frankly of these experiences and accomplishments. This volume will still be of great interest to neurosurgeons in generations to come.

DEAN H. ECHOLS, M.D.

NOTICES

AMERICAN BOARD OF NEUROLOGICAL SURGERY

The following specialists were certified at a meeting of the American Board of Neurological Surgery in Chicago in June 1950.

ADAMS, JOHN EDWIN University of California Hospital, San Francisco 22, Calif.
BAKODY, JOHN T. 900 Des Moines Bldg., Des Moines 9, Ia.
DREW, JOHN HOWARD Cushing V.A. Hospital, Framingham, Mass.
FAUSSET, C. BASIL 408 Hume Mansur Bldg., Indianapolis 4, Ind.
FOWLER, WILLIAM J. 901 Vancouver Block, Vancouver, B. C., Canada
GARCIA-BENGOCCHEA, FRANCISCO 1430 Tulane Ave., New Orleans, La.
GRIFFIN, JOHN GORDON 3500 East 17th Ave., Denver, Colo.
HEIMBURGER, ROBERT F. Indiana University Medical Center, Indianapolis 7, Ind.
KNIGHTON, ROBERT S. Henry Ford Hospital, Detroit 2, Mich.
LEVIN, GRANT 516 Sutter St., San Francisco 2, Calif.
LINDSTROM, PETTER A. 416 North Bedford Drive, Beverly Hills, Calif.
LOWREY, JOHN JEWETT The Clinic, 881 South Hotel St., Honolulu, Hawaii
NEILL, CHARLES LAMAR, JR. 746 Manship St., Jackson, Miss.
OSHER, DANIEL 871 Doctors Bldg., Cincinnati 2, Ohio
POLLARD, CLAUDE, JR. 411 Hermann Professional Bldg., Houston, Tex.
RITCHIE, WALLACE PARKS 917 Lowry Medical Arts Bldg., St. Paul 2, Minn.
RIX, ROBERT ALVIN, JR. 521 N.W. 11th, Oklahoma City 3, Okla.
ROBSON, JOHN THEODORE 1318 Medical Arts Bldg., Tacoma 2, Wash.
SCHNEIDER, RICHARD C. University Hospital, Ann Arbor, Mich.
SIMMONS, DONALD R. 1609 David Broderick Tower, Detroit 26, Mich.
STEELMAN, HENRY F. Wisconsin General Hospital, Madison, Wis.
SUGAR, OSCAR 224 South Michigan Ave., Chicago, Ill.
SVIEN, HENDRIK J. 102–110 Second Ave., S.W., Rochester, Minn.
WALKER, JAMES CHRISTIE Scott and White Clinic, Temple, Tex.
WEBB, EUGENE MCDANIEL 384 Post St., San Francisco 8, Calif.

HARVEY CUSHING SOCIETY

The 18th annual meeting of the Harvey Cushing Society was held in Colorado Springs, Colorado, on April 15, 16 and 17, 1950. Officers elected for 1950–1951 are: President, Dr. W. Edward Chamberlain, Philadelphia; Vice-President, Dr. Harry Wilkins, Oklahoma City; Secretary-Treasurer, Dr. Louise Eisenhardt, New Haven.

Members of the Executive Committee are: Dr. Franc D. Ingraham, Boston; Dr. Frank R. Teachenor, Kansas City; Dr. Barnes Woodhall, Durham; Dr. Winchell McK. Craig, Rochester, Minn., and Dr. Frank Turnbull, Vancouver, B. C.

Dr. Leonard T. Furlow, St. Louis, was appointed to the American Board of Neurological Surgery.

SCANDINAVIAN NEUROSURGICAL SOCIETY

The 6th meeting of the Scandinavian Neurosurgical Society will take place in Lund, Sweden, August 28 and 29, 1950. Members of American and other European neurosurgical societies are cordially invited to attend. Information is obtainable from the Secretary, Dr. Olof Sjöqvist, Södersjukhuset, Stockholm, Sweden, or from the host of the meeting, Dr. Nils Lundberg, Neurosurgical Department, Lasarettet, Lund, Sweden.

A NEW ABSORBABLE HEMOSTATIC BONE WAX

PRELIMINARY REPORT*

JOHN R. GEARY, M.D., AND VIRGINIA KNEELAND FRANTZ, M.D.
Department of Surgery and the Surgical Pathology Laboratory, College of Physicians and Surgeons, Columbia University, New York, N. Y.

(Received for publication December 10, 1949)

THE experimental studies that led to the development of an absorbable and specifically hemostatic bone wax for use in various fields of surgery are described in a paper that has been submitted for publication elsewhere.[1] As bone wax, however, is a special hemostatic device introduced early in the history of neurosurgery by one of its pioneers, it would seem of interest to present this new material in a neurosurgical journal.

It will be remembered that in 1885, on the threshold of a brilliant surgical career, Victor Horsley was conducting experiments in one of the laboratories of the University of London. Acting on a hint obtained from Magendie's *Physiologie*, where a method of stopping sinuses with wax is described, he smeared the bleeding cut surfaces of the cranial bones with modelling wax. He was gratified to discover that the bleeding was instantly and easily controlled. Subsequent to these experiments, a formula was worked out which gave a product having the desired physical properties for use in neurosurgery. The material was kept in stoppered bottles and sterilized by boiling. In response to an inquiry about his wax, Horsely wrote a short note in 1892 in the British Medical Journal describing the formula as follows: beeswax 7 parts; almond oil 1 part; salicylic acid 1%.[3]

Ordinary bone wax as used by the surgeon today is effective in controlling hemorrhage from the cut surfaces of bone only by virtue of its action as a tamponade. It possesses no inherent hemostatic qualities of its own. In addition, it is not soluble in the body fluids and thus remains at the site of implantation for long periods of time. That portion of it which eventually is removed is probably carried away through the action of phagocytic cells. As a result, the wax acts as a foreign body tending to promote infection and in itself causing a low-grade inflammatory reaction in the tissues about the site of implantation.

The new bone wax is specifically hemostatic by virtue of the incorporation of oxidized cellulose in the base. Oxidized cellulose has already had extensive clinical trial in neurosurgery in the form of cotton and gauze.[4] The base chosen however, at the suggestion of Henderson,[2] is not beeswax but carbowax, which is water soluble and has been shown to be non-toxic and

* The work described in this paper was carried out under a grant from the Johnson and Johnson Research Foundation. The experimental materials used in the study were supplied by the Department of Clinical Research, Ethicon Suture Division, Johnson and Johnson, New Brunswick, New Jersey.

non-irritating. Our own experimental studies have demonstrated that it is rapidly absorbed both from soft parts and bone, leaving no residuum.

The first 100 consecutive cases in which it was given clinical trial at the Neurological Institute of New York break down as follows:

Differential section of trigeminal nerve	11
Prefrontal leukotomy	7
Trephination of skull	5
Craniotomy	39
Suboccipital craniectomy	4
Topectomy	6
Laminectomy	16
Interlaminar removal of herniated nucleus pulposus	12
Total	100

Since these first 100 cases, no further detailed records have been analyzed. The material, whenever available, has been used routinely in preference to ordinary bone wax. Technical difficulties in making a uniform product of optimum malleability have somewhat limited the amount available for clinical trial. The wax can be sterilized by autoclave and easily rinsed off operating room gloves because it is water soluble.

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CONSERVATIVE SURGICAL THERAPY OF BRAIN ABSCESSES*

LESTER A. MOUNT, M.D.

Department of Neurosurgery, College of Physicians and Surgeons, and the Neurological Institute, New York, N. Y.

(Received for publication December 13, 1949)

IN THESE days when the fashion in the therapy of brain abscess is complete removal, it is pertinent to call attention to a conservative method of surgical therapy. The best results yet reported are those of Macewen,[6] who in 1893 advised drainage with a small tube. His patients all had chronic abscesses and 18 of the 19 recovered. Without discussing the modification in this type of drainage, as described by Lemaitre,[5] Dowman,[3] Grant,[4] Coleman,[1,2] and others, I will describe the method here reported, present illustrative cases and give the results in the cases so far treated.

The method consists first of accurate localization of the abscess, using ventriculography if necessary, making a trephine opening directly over the abscess, opening the dura, cauterizing the exposed arachnoid and pia so as to seal off the subarachnoid space and prevent meningitis, inserting a needle into the abscess, removing only enough pus for culture and smear, and removal of the needle, insertion of a size 10 catheter with multiple openings at the end, carefully washing the contents of the abscess cavity with penicillin until the return is clear and filling the cavity of the abscess with penicillin, and complete closure of the wound. The catheter is sutured to the scalp and is brought out through the dressing in order to continue the irrigation of the abscess cavity and further instillation of the proper anti-biotic as determined by the culture and smear of the pus removed. The abscess cavity is irrigated every 6 hours until the return is clear and then penicillin is allowed to run into the abscess cavity by gravity in order to completely inflate the cavity and prevent loculation of pus. If the anti-biotic will not run in by gravity a lumbar puncture is done at the time in order to expand the abscess cavity and hence fill it with the anti-biotic. This process is continued until the abscess cavity has completely disappeared and no more anti-biotic runs into the cavity by gravity. The catheter is then withdrawn 1 cm. every other day until it is completely removed. This requires an average of 3 weeks from the time of operation, but with larger abscesses more time is required. In addition to the local treatment, the patient is given the proper anti-biotic intramuscularly or by mouth.

ILLUSTRATIVE CASES

Case 1. R.F. #811411. This was the first Neurological Institute admission of a 54-year-old white married female, transferred from the Presbyterian Hospital. The

* Presented before the New York Neurological Society, March 8, 1949.

history was obtained from her daughter. The patient had been in good health until about 7 weeks before, when the daughter believed she had a cold. A week later she complained of pain in her left ear and a few days later an abscess opened by itself. A local doctor advised irrigation with saline solution. Two weeks later the pus ceased draining but 4 weeks later it began to drain again. She did not improve with sulfa drugs and was admitted to the Presbyterian Hospital for mastoidectomy. Two days before admission there, she began to have difficulty in finding the right words when speaking. This suddenly became worse and then remained unchanged. Two days before her mastoid operation she began to complain of pain in the top and back of the head. The day after the mastoidectomy she was seen by a neurosurgical consultant, who found nominal aphasia and right central facial paresis, but no papilledema. She was afebrile. During her hospitalization at the Presbyterian Hospital she was treated with 20,000–25,000 units of penicillin every 3 hours. Three weeks after mastoidectomy she was transferred to the Neurological Institute with a diagnosis of brain abscess.

Examination. Her gait was very unsteady. She took small steps and bilateral spasticity was present. There was slight impairment of strength in the right extremities, a little more marked in the upper. Coordination tests were well performed. There was a nominal aphasia. Deep tendon reflexes were more active on the right than on the left. The patient reacted to pin prick throughout but the aphasia precluded a satisfactory sensory examination. There was no papilledema but there was slight enlargement of the retinal veins. The right pupil was 1 mm. in diameter, the left 3 mm. There was a right central paresis.

CSF pressure was 150 mm. of water; fluid was clear; there were 36 wbc., 92 per cent of which were lymphocytes; total protein was 70 mg. per cent. X-ray of the skull was non-contributory. Ventriculography showed the abscess to be in the anterior portion of the dominant temporal lobe.

Operation. A trephine opening was made and a needle was inserted into the lower portion of the middle temporal gyrus. The abscess was encountered at a depth of 1.5 cm. below the surface of the brain. The wall of the abscess was thick and it was with some difficulty that the needle could be pushed through it. Thick, brown-yellow pus was recovered; a total of 20 cc. was removed. The median wall of the abscess was reached at a depth of 4 cm. from the surface of the brain, indicating the diameter of the abscess to be 2.5 cm. The cavity of the abscess was irrigated with penicillin, 500 units per cc., until the return was clear and then 18 cc. of penicillin were allowed to remain within the cavity. A catheter was placed within the cavity for the purpose of further drainage and irrigation with penicillin.

Course. The cavity was irrigated daily and penicillin inserted, the amount depending upon the quantity which could run in by gravity. When the cavity was obliterated the catheter was removed at the rate of 1 cm. daily; it was completely removed on the 20th postoperative day. Postoperatively 25,000 units of penicillin were given intramuscularly every 3 hours. The culture of the abscess showed no growth. The patient was discharged on her 25th postoperative day. When last examined, 14 months after operation, she was asymptomatic. Neurological findings were normal except for a very occasional difficulty in finding the right word.

Case 2. V.H. #859563. The history was obtained from the father, who was not fully informed of the details of the illness. The patient, a girl aged 12, contracted a head cold 2 months before admission and a few days later complained of a right ear

ache. This lasted 1 week. Six weeks before admission there developed right frontal headache and pain in the right eyeball. These symptoms increased in severity and 3 weeks later she began to vomit; she felt chilly but not feverish. The following week she was admitted to the South Hampton Hospital; she had no fever; wbc. was 17,600 with 95 per cent polys. CSF showed 50 wbc. X-rays of the skull were suggestive of "subacute mastoiditis." She continued to vomit, was given parenteral fluids, and penicillin. She seemed to improve and 10 days after admission was discharged as "cured." Her only complaint at that time was generalized weakness. The day after discharge she complained of generalized headache and vomited several times. The headaches became more severe and became localized over the left forehead and behind the left eyeball. The patient became weaker and drowsy and was returned to the South Hampton Hospital. Lumbar puncture showed a pressure of 230 mm. of water and 5 wbc. She was then transferred to the Neurological Institute.

Examination. She was an emaciated, acutely ill girl, who was stuporous. Temperature 100°, pulse 88, respirations 20 and BP 128/78. There was tenderness over the left mastoid process. There was generalized weakness but there was definitely more weakness in the right extremities. There was ataxia, bilateral, on performance of finger-to-nose, and heel-to-knee tests. The deep tendon reflexes in the upper extremities were hyperactive, with right-sided preponderance; the lower extremity reflexes were hyperactive with transient suprapatellar clonus bilaterally. There was a positive Babinski and Chaddock on the right and moderate photophobia and nuchal rigidity. There were 3 D. of papilledema on the right and 4 on the left, with flame-shaped hemorrhages. The pupils were 5 mm. in diameter and reacted well to light and accommodation. There was slight bilateral external rectus paresis. There was seropurulent secretion in both ear canals. Needle exploration was made in the left temporal lobe without finding an abscess.

Ventriculography showed an internal hydrocephalus with a dilated aqueduct shifted toward the left side, indicating a right cerebellar mass.

Operation. Under local anaesthesia trephine opening was made in the right lateral suboccipital region. The dura was opened and a needle inserted. An abscess was reached at a depth of 1.2 cm. The needle was withdrawn and a catheter inserted. A total of 15 cc. of creamy, inspissated pus was removed. The abscess cavity was irrigated with penicillin solution until the return was clear, and penicillin was allowed to remain within the cavity.

Course. The patient's vital signs immediately improved to a marked degree. In fact, for the first time, she began to speak rationally. Culture of the abscess showed pneumococcus Type III. The cavity was irrigated daily until it was completely obliterated. The catheter was gradually removed, complete removal being accomplished on the 28th postoperative day. The patient was discharged on the 36th postoperative day. The papilledema had subsided. There was some unsteadiness when standing on either foot alone but otherwise the neurological findings were negative.

Two weeks later she was admitted to the Nose and Throat Service for a mastoidectomy. She made an excellent recovery from this.

Case 3. M.D. #850463. The patient was a white female, aged 1 year. Five weeks before admission, hoarseness, severe dry cough, and vomiting developed. A week later she began to have fever, sometimes as high as 104°. She lost her appetite, and became listless and irritable. She was treated with penicillin, sulfadiazine, and streptomycin, without complete remission of fever, and the symptoms persisted. A few

days before admission photophobia developed. A lumbar puncture was done. There were 1,300 wbc., 82 per cent of which were polys; sugar was 33 mg. per cent; no organisms were present on smear, and culture was negative. There had never been a stiff neck.

Examination. She was thin, pale, irritable, and drawn. She was able to sit and stand and was apparently responsive to her environment. The anterior fontanelle was soft; there was no crackpot sound. There was some resistance to flexion of the neck. There were palpable cervical, axillary and inguinal lymph nodes. The deep tendon reflexes were all equal and hyperactive. There was no Brudzinski, Kernig, or Babinski.

Lumbar puncture showed cloudy fluid, 1,310 wbc.; sugar 20 mg. per cent; culture Hemophilis influenzae, Type B. X-rays of chest and skull were negative.

Course. After a short course of sulfadiazine by mouth the patient was given sulfadiazine parenterally and streptomycin intrathecally every day for 3 days, as well as streptomycin intramuscularly for 3 days. Sulfadiazine parenterally was continued for 10 days. Because the organism was found to be incompletely susceptible to streptomycin, antiserum was given. The patient did not respond well to therapy and began running an irregular temperature up to 103° in the 4th week after admission, and the white cells in the fluid were consistently high up to 17,500. There was now a sustained ankle clonus on the right and a right Babinski. X-rays of the skull showed all the sutures to be widened. A pneumo-encephalogram revealed a large space-occupying mass on the left side supratentorially.

Operation. A trephine opening was made in the left posterior parietal region and thick greenish pus was found in the subdural space; 80 cc. were removed. A rubber catheter was inserted into the subdural space and 10 cc. of thorotrast were inserted into the abscess cavity. X-rays were taken and these showed the abscess to cover the entire left hemisphere. The thorotrast was then washed from the abscess cavity and the cavity was filled with a solution of streptomycin-sulfadiazine. Culture of the abscess showed Hemophilis influenzae, Type B.

Course. The cavity was irrigated twice daily and the same solution allowed to remain within the cavity; in order to keep the subdural abscess open, lumbar puncture was performed with drainage of the fluid at the time the cavity was irrigated. She progressively improved on this regime and from the 3rd postoperative day until the time of discharge, 4 weeks later, she was completely afebrile. She gradually became more cheerful, alert and talkative. The abscess cavity gradually closed. The catheter was removed 17 days after operation. The patient was discharged on the 35th day after operation. There were no positive neurological findings except for hyperactive deep tendon reflexes. The anterior fontanelle was small and depressed. When last seen, 15 months after operation, she was asymptomatic.

COMMENT

Ten patients with brain abscesses, 2 cerebellar, 1 subdural and the rest cerebral, have now been operated upon by this method, with recovery in 8 cases. In 1 of the unsuccessful cases the abscess had already ruptured into the ventricle at the time of operation; and in the other, drainage was not adequate by the catheter method and the abscess was treated by marsupialization the day after the catheter method of therapy was attempted. In the 31 cases of brain abscesses treated at the Neurological Institute during the

same period, but by other methods, the mortality rate was 45 per cent. The catheter method of treatment affords drainage of the abscess with a minimum amount of cerebral trauma. This is especially valuable when the abscess extends into important areas of the brain, such as the motor or speech areas. The catheter method cannot be used in every case of abscess. If free exchange of fluid through the catheter cannot be obtained, this method of therapy is inadvisable. It is important that the proper anti-biotic be used. This, of course, is determined by testing the sensitivity of the organism recovered from the pus to penicillin, streptomycin, and bacitracin.

SUMMARY

A conservative method of treatment of intracranial abscesses was described. The method consisted of accurate localization of the abscess, trephination over the abscess cavity, drainage of the abscess, and the use of antibiotics in the abscess cavity, and systemically. The mortality rate was 20 per cent. In the abscess cases treated at the Neurological Institute by other methods during the same period the mortality rate was 45 per cent. This method of therapy cannot be used in the treatment of every intracranial abscess.

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PRECENTRAL MOTOR CORTICAL ABLATION IN EXPERIMENTAL EPILEPSY IN THE MONKEY

B. L. PACELLA, M.D., M. A. KENNARD, M.D.,* L. M. KOPELOFF, PH.D., J. G. CHUSID, M.D., AND N. KOPELOFF, PH.D.

Department of Bacteriology, Psychiatric Institute, the Departments of Anatomy and Psychiatry, New York University Medical School, and the Neurological Division, St. Vincent's Hospital, New York, N. Y.

(Received for publication December 16, 1949)

THE application of alumina cream to the precentral motor cortex has consistently induced recurrent convulsive seizures and electroencephalographic (EEG) abnormalities in the monkey.[1] Subtotal removal of areas 4 and 6 including the cortex underlying the disc resulted in the cessation of seizures for a period of 1 to 2 years, and considerable diminution in the EEG abnormality.[3] Subsequently, in one of the monkeys described the EEG disturbance returned to its former pre-ablation pattern. A more extensive ablation of the surrounding cortical tissue was then performed, but the abnormality persisted and was followed later by a return of seizures.

These findings suggested the need for further investigation. The present study includes continued observation of the previously described animals, as well as a more extensive series of ablations performed *prior* to the application of alumina cream. In some instances the alumina was placed in contact with the subcortical tissue underlying an ablated area, while in other cases it was applied to an adjacent intact cortical site.

EXPERIMENTAL

Cortical ablations were performed under pentobarbital sodium anesthesia. A bone flap was made at the appropriate region. The dura was then reflected and the cortical area removed by gentle suction, the blood vessels being coagulated or closed with silver clips. Closure was made at dural, muscle, and skin levels.

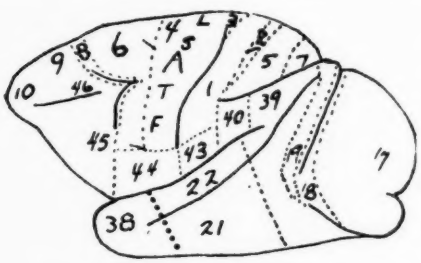

FIG. 1. Cortical map of *Macaca mulatta* after McCulloch.[2] F—face, T—thumb, A—arm, S—shoulder, L—leg.

In 4 of the 6 monkeys in which bilateral ablations were performed an interval of 1 month was allowed to elapse between operations. Alumina cream was then applied at a third operation on the side of the first ablation.

EEGs were recorded at intervals during the observation period.[3] At the termination of the experiment all ani-

* Now at the Department of Surgery, University of Oregon Medical School, Portland, Oregon.

mals were sacrificed for anatomical study. Individual analyses and diagrams showing the areas ablated and sites of application of alumina cream were made with the aid of a cortical map of *Macaca mulatta* similar to that used by McCulloch[2] (Fig. 1).

SCHEMA

I. *Ablation Prior to Application of Alumina Cream.* In 11 monkeys cortical ablations were performed 3 to 6 months prior to the application of alumina cream. These included subtotal removal of various areas in the precentral motor region of

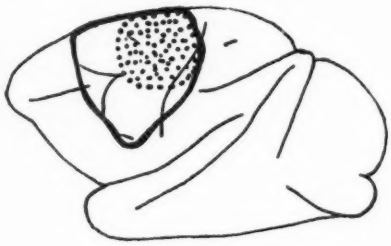

FIG. 2a. #487.

FIGS. 2–8. The heavy solid outline indicates the cortical area ablated, and the dotted portion the site of the disc containing alumina cream.

In Figs. 2b, 2d, the arrow indicates the site of injection of alumina cream. In Figs. 6, 8, the probable site occupied by the disc before its removal at the time of ablation is indicated by crosses.

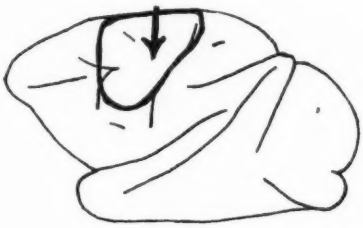

FIG. 2b. #513.

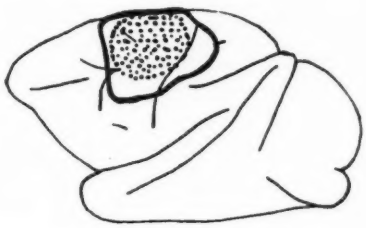

FIG. 2c. #514.

FIG. 2d. #512.

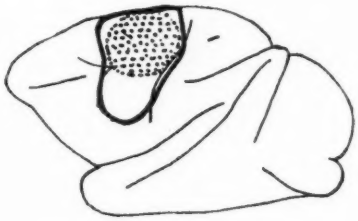

FIG. 2e. #515.

one or both cerebral hemispheres. In no instance was the motor cortex of the mesial aspect (4, 6, foot) ablated. The principal areas removed included the following:

(A) Unilateral areas 4 and 6 followed by alumina cream placed in disc on (#487, #514, #515) or injected into (#513, #512) the ablated site (Fig 2).

Extensive ablations of left 4 and 6 on the convexity. Areas 6 and 4 in their most lateral (face) portions were sometimes not completely ablated. Adjacent areas an-

teriorly and posteriorly were sometimes included (8, 46, 45, and 3–1–2). The most extensive ablations were performed in #487 and #515. The deepest ablations were made in #512 and #515.

(B) Bilateral areas 4 followed by disc of alumina cream over area 6 of one side (Fig. 3).

Relatively good 4 ablations bilaterally. Areas 6 laterally (face) included in #521 and to a minimal degree on the right in #538.

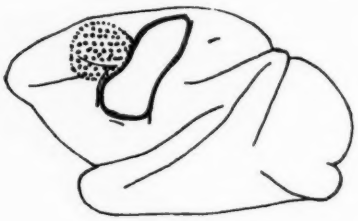

FIG. 3a. (L) #521.

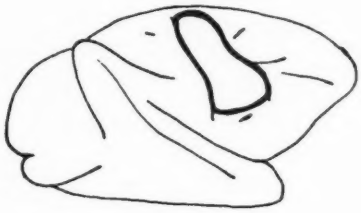

FIG. 3a. (R) #521.

FIG. 3b. (L) #538.

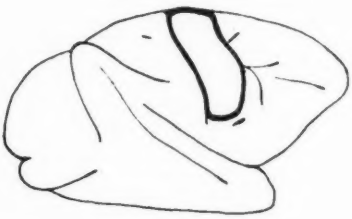

FIG. 3b. (R) #538.

(C) Area 4 of one side and area 6 of the opposite side with application of alumina cream disc over area 6 adjacent to the area 4 ablation (Fig. 4). On left: good 4 ablation including 6, face. On right: ablation of 6 and small portion of adjacent 4. The 6 ablation was incomplete because of failure to include 6 face and probably 6 thumb.

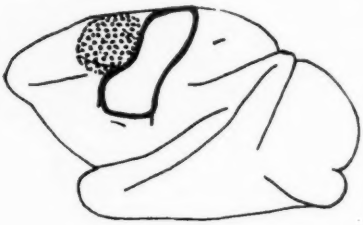

FIG. 4. (L) #508.

FIG. 4. (R) #508.

(D) Bilateral areas 6 followed by alumina cream disc over area 4 and 3–1–2 of one side (Fig. 5). In these animals there was partial ablation of 6 with preservation of 6 face and 6 thumb bilaterally.

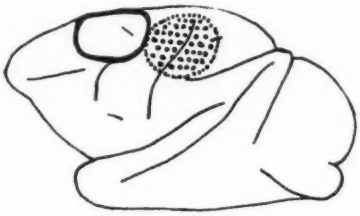

FIG. 5a. (L) #536.

FIG. 5a. (R) #536.

FIG. 5b. (L) #528.

FIG. 5b. (R) #528.

II. *Ablation Following the Development of Recurrent Seizures.* In 3 monkeys the ablation of cortical tissue was performed on the same side of the brain as the original application of alumina cream. The motor cortex of the mesial aspect of the ipsilateral side was left intact.

(A) Alumina applied over left motor area followed by ablation of left areas 4 and 6. The face area of 4 and 6 was not completely removed (Fig. 6).

(B) Alumina applied over left motor area followed by ablation of left treated site.

(C) Alumina applied over left area 4 followed by partial ablation of left area 6. The portion of area 6 lateral to the angle of the arcuate sulcus (thumb, face) was left intact (Fig. 7).

FIG. 6. #452.

III. *Ablation After Treatment of Cortex with Inactive Material.*

(D) In one monkey a disc of zinc oxide was applied over the left motor cortex, followed by subsequent ablation of the treated site including portions of areas 4, 6, 8, and 3–1–2 (Fig. 8).

OBSERVATIONS

Ablation Prior to the Application of Alumina Cream. In Table 1 are summarized the results obtained when ablation preceded the application of alumina cream. Seizures developed in 9 of the 11 monkeys of this group and in 7 of these the attacks later became bilateral. EEG abnormalities were

noted in all but 1 of the convulsing monkeys. In the 2 monkeys that failed to react, the removal of tissue was greatest and extended almost to the surface of the lateral ventricle at several points. One (#512) received alumina by injection, while in the other (#515) a disc of alumina cream was applied to the ablation site. In all monkeys the presence of small residual areas of

FIG. 7. #504.

FIG. 8. #430.

cortical gray substance in the deeper layers was established by postmortem examination of serial coronal sections at 4 mm. intervals through the ablated sites.

TABLE 1

The effect of cortical ablation prior to the application of alumina cream

Principal Areas Ablated	Monkey Number	Alumina Cream L	Site of Disc	Areas Ablated	Seizures			Maximal EEG Abnormality	
		Mos. After Ablation*			Onset Mos.	Maximal Response†	Duration Mos.	L	R
A. L4, 6	487	6	Fig. 2	a	4½	RL	17	++++	++
	513	3½		b‡	5	RL	5	+++	++
	514	3½		c	2½	RL	12	+±	+±
	512	3		d‡		0		+	+
	515	3½		e		0		0	0
B. LR4	521	3	Fig. 3	a	2½	RL	15	++	++
	538	3		b	2½	R	1§		
C. L4, R6	508	3	Fig. 4		2	RL	4	+++±	+++
D. LR6	536	3½	Fig. 5	a	8	R	4	0	0
	528‖	3		b	2	RL	5	+±	+±
	529‖	3	—		2½	RL	3	+++	+++

* Months after second ablation in groups B, C, D.
† R—right unilateral seizures only; RL—bilateral seizures beginning on right.
‡ Alumina injected beneath ablated site.
§ Died 1 month after onset.
‖ Ablations performed on same day.

In group A the removal of extensive portions of the precentral motor cortex of one hemisphere (Fig. 2) failed to inhibit the development of seizures in 3 of 5 animals. It should be noted that in #487 the ablation was relatively shallow, while in #512 and #515 it extended deepest.

In group B the subtotal removal of areas 4 and 6 bilaterally as indicated in Fig. 3 did not prevent the development of seizures following the application of a disc containing alumina cream over the cortex anteriorly adjacent to one ablated site.

In group C (#508) the subtotal ablation of area 4 of one side and area 6 of the opposite side with the application of alumina cream to the cortex anteriorly adjacent to ablated area 4 did not prevent the development of bilateral seizures.

In group D the subtotal removal of areas 6 bilaterally did not inhibit the development of seizures following the application of alumina over the posteriorly adjacent cortex of one hemisphere.

Ablation After the Induction of Seizures. The effect of ablation in monkeys with well-established seizures is shown in Table 2. Three of the 4 monkeys have been referred to previously.[3] The present study includes these data and subsequent observations. It may be seen from Table 2 that seizures were not permanently inhibited when the cortical tissue underlying the disc and portions of adjacent cortex were removed (#452), and that the EEG abnormality was only temporarily diminished (#452, #451). This qualifies our preliminary observations on the same animals which indicated that seizures and EEG abnormalities were inhibited. In monkey #504 in which only partial removal of left area 6 was performed no significant changes in either seizures or EEG abnormality were noted.

In control monkey #430 ablation of the cortical area underlying the disc containing "inactive" substance (zinc oxide) did not introduce any abnormal clinical or significant EEG changes.

As noted in the previous group, serial coronal sections revealed residual gray islands in all ablated sites.

DISCUSSION

Subtotal ablation of the cortex underlying an active disc of alumina cream in 2 epileptic monkeys temporarily inhibited the clinical and EEG convulsive manifestations.[3] After a period of 4 months to 1 year following ablation, EEG abnormalities recurred and in one animal seizures were again elicited 7 months after a second ablation (performed $1\frac{1}{2}$ years after the first ablation). Subtotal ablation of the precentral motor areas on one or both sides of the brain, followed later by the application of an alumina cream disc to the surface of the ablated zone or the adjacent cortex, did not inhibit the subsequent development of seizures or EEG abnormalities, with the exception of #512 and #515 (Table 1, Fig. 2d, e). In these 2 animals in which neither seizures nor characteristic EEG changes were observed, it may be significant that the depth of the ablations was relatively greater.

TABLE 2

The effect of cortical ablation on convulsive seizures induced by alumina cream

Monkey 452					*Monkey 451**				
Alumina L Motor	Seizures Before Ablation		EEG Abnormality		Alumina E Motor	Seizures Before Ablation		EEG Abnormality	
	Type	Duration Mos.	L	R		Type	Duration Mos.	L	R
	RL	5½	+++	++		RL	5½	+++ ++	++± +
Months After Ablation	Seizures After Ablation				Months After Ablation	Seizures After Ablation			
	L4					L4*			
2	0		+	±	½	RL		++	+
4	0		+++	++	1	0		+	+
18	0		++±	++	2	RL		+	±
	L4, 6 (Fig. 6)				7	0		0	0
1	0		+++	++	9	0		++	±
3	0		+++	++	12	0		+++	+++
6½	R		0	0	13	0		+++	++++
7½	L								
9	LR								
10	0		++	±					
13	0		+++	+					
19	LR								
Monkey 504					*Monkey 430**				
Alumina L Motor	Seizures Before Ablation		EEG Abnormality		Zinc Oxide L Motor	Seizures Before Ablation		EEG Abnormality	
	Type	Duration Mos.	L	R		Type	Duration Mos.	L	R
	RL	12	++± +	+ +		0	16	0	0
Months After Ablation	Seizures After Ablation				Months After Ablation	Seizures After Ablation			
	L6 (Fig. 7)					L4, 6* (Fig. 8)			
2	RL		+±	0	2	0		0	0
6	RL		+++	++	10	0		0	0
16	RL		+	0	17	0		0	0
					39	0		0	0

* Ablation performed by electrocauterization.

The presence of residual gray substance in all ablated areas was a constant finding at postmortem examination. Whether these remnants were potentially epileptogenic can neither be established nor excluded. However,

it is noteworthy that clinical and EEG abnormalities did develop in the absence of relatively large masses of precentral motor cortex, and furthermore could not be distinguished from those found in unablated epileptic monkeys. The possibility exists that subcortical tissue or circuits may function in the production of the epileptic abnormalities described either primarily, or secondarily in the absence of cortex.

Spread of alumina cream to unablated cortex from its site of application should be considered. If this occurs it is possible that the epileptic manifestations observed resulted from action on adjacent cortex rather than on the presenting subcortical tissue or residual gray matter of the ablated site. However, since the clinical pattern of seizures was not stereotyped, and individual attacks did not always begin with foot or leg movements, it may be assumed that, in some instances at least, the action of alumina was not on the remaining unablated foot-leg cortical area.

SUMMARY

1. Subtotal ablation of the precentral motor cortex on one or both sides did not inhibit the subsequent development of recurrent convulsive seizures or EEG abnormalities following the application of alumina cream to an ablated site or the adjacent cortex in *Macaca mulatta*.

2. In 2 additional monkeys with well-established seizures the ablation of cortical tissue underlying the alumina cream disc, and including portions of adjacent cortex, caused only a temporary diminution of EEG abnormality and suppression of clinical seizures for 7 months and 1 year respectively. Nevertheless, in the former, seizures recurred for 13 months thereafter.

3. It was noted that in the ablated sites small residual areas of gray substance were present at postmortem examination. The implications of this finding are discussed.

We wish to thank Frances Simpson for her assistance in recording electroencephalograms.

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ATROPINE IN THE TREATMENT OF CLOSED HEAD INJURY

ARTHUR WARD, JR., M.D.

Neurosurgical Service, King County Hospital, and Division of Neurosurgery, University of Washington School of Medicine, Seattle, Washington

(Received for publication December 21, 1949)

THE accepted treatment of severe closed head injury is largely concerned with supportive measures,[15] and no definitive therapy has been described that is directed at any specific reversal of the non-surgical pathophysiologic sequelae of trauma of the brain.

Information has slowly accumulated regarding the mechanism of concussion,[3] including descriptions of the pathologic changes such as those described by Scheinker[12,13,14] as "vasoparesis." Studies regarding cerebral swelling have been controversial,[11] and treatment by dehydration has now been largely discarded. On the basis of present knowledge, it would seem that prolonged coma is due, at least in part, either to unknown changes in the neurone or to alterations in the environment of the nerve cell which persist for long periods of time.

This would suggest the possibility that metabolic changes may occur in neurones as the result of trauma. Studies of this type have been carried out only in recent years and only a small amount of data is available. Gurdjian, Webster and Stone[6] have studied the carbohydrate metabolism of damaged brain and have demonstrated that head injury in animals causes no significant changes in the cerebral arteriovenous differences in oxygen, carbon dioxide, and glucose. Specimens of cerebral tissue were obtained by freezing the brain *in situ*, and it was found that areas of contusion showed greatly increased lactic acid and inorganic phosphate with decreased phosphocreatine and adenosine triphosphate. They felt that these findings might be due to a combination of direct injury to cells and anoxia resulting from vascular damage. However, areas of cortex showing no macroscopic evidences of damage were often chemically normal, even in profoundly injured animals. Thus, it is evident that head injury causes no generalized disturbance in cerebral oxidations.

However, changes in acetylcholine metabolism do exist following brain trauma. Bornstein[1] has pointed out that while free acetylcholine (ACh) is never normally present in cerebrospinal fluid, it is found in relatively large quantities shortly after trauma in the experimental animal. It appears that ACh is liberated in abnormal amounts by traumatized nervous tissue and that some of the liberated ACh escapes destruction, persists in the intercellular spaces and finally diffuses into the CSF where its presence may be quantitatively determined. He also felt that there was a positive correlation between the concentration of ACh in the CSF, the clinical signs of concussion and the post-concussion EEG changes. He concluded that "free ACh

may be one of the physiological factors underlying the acute paralytic and excitatory phenomena of cerebral concussion and more severe craniocerebral injuries." Bornstein further reasoned that if the abnormal electrical activity of the cortex and the behavioral pattern following concussion are due, at least in part, to the activity of abnormal concentrations of ACh within the brain tissue, it should then be possible to reverse these effects by atropine, an anticholinergic drug. He found this to be the case in the experimental animal in that both the EEG patterns and the stuporous condition may be abolished by appropriate doses of atropine sulfate (0.5–1 mg. per kg.). Bornstein also demonstrated that intracisternal injection of ACh (0.02 to 10 gamma) produces behavioral and EEG changes similar to those noted following concussion, and these may also be abolished by atropine. Tower and McEachern[16] have studied these factors following head injury in the human and found low cholinesterase activity with reversal of normal cholinesterase ratios as well as free ACh, which may be present in large amounts in severe cases. Recovery is associated with reversal of the above changes. In 3 patients they could demonstrate a correlation between cholinesterase pattern, acetylcholine level, EEG, and the clinical state of the patients. It is obvious that these findings clearly suggest a rationale of therapy specifically directed at the reversal of one of the pathophysiologic sequelae of trauma of the brain.

CLINICAL DATA

Since it is difficult to accurately forecast the clinical course of patients who have been subjected to relatively mild head injury, anticholinergic treatment was given initially only to those patients who obviously had sustained very severe brain trauma. All of the initial group of 20 patients had severely damaged brains as evidenced by depth of coma, focal neurological signs and grossly bloody CSF. It was recognized that anatomical damage to the CNS was present in these cases and that no physiological methods could reverse these changes; but since superimposed reversible physiological factors should also be playing a role, it was hoped that obvious and reproducible clinical improvement could be produced in certain cases by the administration of atropine. The atropine sulfate was administered subcutaneously in doses of 0.1 mg. per kg. of body weight, so that the average single adult dose was gr. 1/10.

In one of the most striking cases of this group, the clinical improvement could be directly related to the prior administration of atropine, and this was consistently reproducible over 5 cycles. The patient was initially in decerebrate rigidity and each cycle consisted of dramatic clinical improvement which became definite about 12 to 14 hours after atropine. This was maintained for about 10 hours or slightly longer. The state of consciousness would then slowly become depressed over a 24 to 48 hour period, approaching the original level, at which time atropine was again administered. This was one of the early cases treated in this fashion, and for that reason the rather

large doses of atropine were given at rather cautious intervals.

Of the remaining 19 patients, 2 exhibited typical decerebrate rigidity on admission and both ultimately recovered. There were 5 fatalities in this group, and gross brain damage was present at autopsy in all instances. Of the 15 patients who recovered, it was felt that anticholinergic therapy was of dramatic benefit in 4 cases, of definite benefit in 7 cases, and of questionable value in 4 cases. Because of the satisfactory clinical results in this initial series of severely injured patients, anticholinergic therapy has been used in an additional series of 72 cases. These cases represented routine instances of closed head injury in patients admitted to the Neurosurgical Service in whom no improvement in the state of consciousness was noted during the first 3 hours after admission. In many of these the average adult dose of gr. 1/10 was administered daily as long as the patient was unconscious. Since the therapeutic benefit is more difficult to assess in such an unselected group of cases, no conclusions can be drawn in a small series other than the crude clinical impression that certain (but not all) patients recovered from the effects of trauma with greater rapidity than would otherwise be anticipated.

DISCUSSION

The ideal group of cases for a study such as this would be one in which the brain had been subjected to maximal "concussive" trauma without evidence of any gross brain damage whatsoever. Obviously this was not the case in any of the initial cases reported. Even so, anticholinergic therapy was of benefit in a significant number. This might tend to substantiate the conclusion of Denny-Brown and Russell[4] that "there is often no evidence to show that contusion prolongs the concussion disturbance of function." However, in those cases that were ultimately fatal, adequate pathological explanation of the cause of death was present. Obviously such changes cannot be reversed by anticholinergic therapy.

The mechanism by which acetylcholine is liberated by the mechanical energy of concussion remains unknown. Lorente de Nó[9] has shown that ACh is released from peripheral ganglia by mechanical damage alone. It is also conceivable that the observed free ACh might be the result of the intense neuronal discharge described by Walker, Kollros and Case[17] and Bornstein[1] suggested that this factor might be responsible for the presence of free ACh in his experimental animals. This is perhaps still open to question in view of the observation of Williams and Denny-Brown[19] that blows resulting in concussion always caused an instantaneous and generalized diminution in the cerebral electrical activity rather than hyperactivity, although blocking of amplifiers prevented recording during the first few seconds after trauma. It is known that cholinergic drugs injected into the subarachnoid space will cause many clinical signs similar to those seen in concussion, and these effects may be prevented by parenteral administration of atropine.[7,8] There is also some evidence that cholinergic compounds in

adequate concentrations will cause hemorrhages in the grey matter accompanied by diffuse gliosis, and subsequently by development of glial nodules, glial scars and neurone depletion.[2] These observations certainly do not disallow the concept that the presence of excess amounts of free ACh may contribute to clinical signs of head injury. We know that ACh is a powerful vasodilator and will cause increased permeability of the blood-brain barrier[5] and thus certain of the pathological changes described by Scheinker[12] may be on this basis. Since ACh may play some role in synaptic transmission, this disturbance of neuronal metabolism may also account for a part of the depression of neuronal activity seen clinically.

The dosage used in these series of cases was approximately 0.1 mg./kg., while Bornstein[1] used 0.5–1.0 mg./kg. in his experimental animals. On that basis, the human dose used should be considered minimal to produce the necessary anticholinergic effect. However, it was felt that a single dose of gr. 1/10 every 24 hours in these cases was close to toxic levels. In about 25 per cent of all cases some definite change in heart rate was observed, the pupils often became moderately dilated, there was a variable increase in temperature, and rarely a cutaneous flush was seen. In all instances these signs regressed within 4 to 6 hours and were no cause for concern. Because of the action of this drug on the heart, anticholinergic therapy has not been used in patients with proven cardiac disease. Sporadic reports of atropine poisoning appear in the literature,[10,18] but it is evident that this drug possesses a surprising factor of safety.

It is obvious that atropine does not represent the ideal anticholinergic drug for this particular purpose, and the search for a more satisfactory substitute is under way. In this study it would have been helpful, both in the matter of determining dosage and also in assessing the result of treatment, to have had serial EEGs on each patient. That was not done in the present series, but will be done in a future series.

Although certain very dramatic results have been achieved by anticholinergic therapy in patients with head injury, it is somewhat disappointing that unquestionable improvement due to medication alone could not be demonstrated in approximately 20 per cent of the cases. It is easy to hypothecate that the factor of organic damage was responsible for these doubtful cases. However, the essential fact is that we do not have sufficient knowledge to understand all the details of the pathophysiology of cerebral trauma and therapy must of necessity be somewhat empirical at present. It is hoped that the search for specific therapy will yield some of the needed data.

SUMMARY

1. It is known that following cerebral trauma, both in experimental animal and in man, free acetylcholine is found in the CSF in rather large amounts, whereas normally none is present. In the experimental animal both the EGG patterns and the stuporous condition resulting from cerebral trauma may be abolished by appropriate doses of atropine sulfate.

2. On the basis of the above data, anticholinergic therapy by atropine sulfate in doses of 0.1 mg./kg. (gr. 1/10 in adult man) has been used in a series of 20 patients who had sustained very severe closed head injuries. Dramatic and consistently reproducible clinical improvement was obtained in selected instances. Because of these favorable results, anticholinergic therapy has been used in an additional series of 72 consecutive patients with all grades of cerebral trauma.

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VENTRICULOCISTERNOSTOMY ACCORDING TO TORKILDSEN

A REPORT OF TWENTY-TWO CASES

LENNART HERLIN, M.D.

Neurosurgical Clinic, Serafimerlasarettet, Stockholm, Sweden*

(Received for publication January 3, 1950)

To RE-ESTABLISH the circulation of the cerebrospinal fluid (CSF) presents difficult problems in cases of hydrocephalus with increased intracranial pressure due to Sylvian aqueductal obstruction or inoperable tumours in or near the 3rd ventricle. A great number of palliative operations have been tried to effect a new passage for the CSF between the ventricular system and the subarachnoidal space, or other tissues and organs of presumed resorptive or conductive capacity. Dandy[2] introduced the procedure of ventriculostomy, establishing a communication between the 3rd ventricle and the cisterna chiasmatis by puncturing the lamina terminalis. This operation, still in use, has the disadvantage that the puncture is performed externally beneath the frontal lobes, thus puncturing also the arachnoidal membrane. There is therefore a risk of CSF escaping subdurally, where the power of absorption is poor. Also, the ventriculostomy often tends to close after some time. Mixter[7] performed ventriculostomy from the inside through the floor of the 3rd ventricle into the cisterna interpeduncularis. In this procedure a cystoscope was passed through one of the lateral ventricles and reached the 3rd ventricle via the foramen of Monro. The floor of the 3rd ventricle was then punctured with a flexible probe. His patient, a 9-month-old child with a non-communicating hydrocephalus, was temporarily much better postoperatively. The general condition improved, the size of the skull diminished, and the dye test showed a communication between the ventricular system and the lumbar subarachnoidal sac. Later, the same idea was used by Stookey and Scarff;[9] in their procedure the lamina terminalis was punctured from the outside as a first step. The floor of the 3rd ventricle was then punctured from within the ventricle, thus connecting the ventricle with the cisterna interpeduncularis, and leaving the arachnoidal membrane intact. The brain tissue punctured in this procedure is very thin, and the authors were of the opinion that there was no fear of a secondary closure.

Hyndman[4] established a communication between one lateral ventricle and the cisterna ambiens by removing the glomus of the choroid plexus. At this clinic Leksell tried essentially the same procedure in a few cases of atresia of the Sylvian aqueduct by puncturing the laminae of the choroid plexus between one lateral ventricle and the cisterna ambiens.

* Chief: Professor H. Olivecrona.

In the Torkildsen[10] procedure a direct communication is established between the body or posterior horn of one lateral ventricle and the cisterna magna by means of a tube, usually of rubber. Both the Torkildsen and the Hyndman procedures can be used in cases of tumour in or near the 3rd ventricle.

Cases suitable for the palliative operations described in the brief review above are not exceptional, but are rare enough to make it impossible for one single clinic to have at its disposal a sufficiently large clinical material to judge reliably the indications for the different operations and their respective values. Most of the publications dealing with this special subject therefore comprise only occasional or few instances. Torkildsen[11] in his monograph published his results in 32 cases. Fincher, Strewler, and Swanson[3] reviewed the literature and reported their results in 19 cases in which the Torkildsen method was used.

CLINICAL MATERIAL

At this clinic 31 patients have been operated upon by Torkildsen's method during the years 1939 to 1948. Nine of them had undergone other

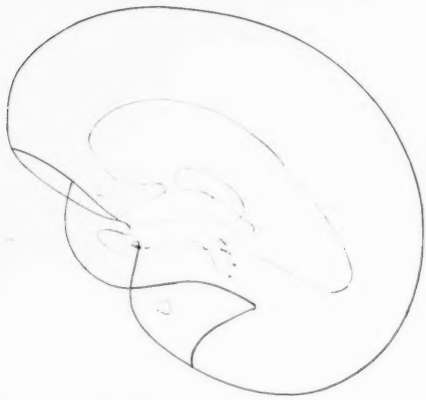


FIG. 1. Case 6. Lateral view of ventriculogram (diagram according to Lysholm).[6] The lateral ventricles are symmetrically dilated. The 3rd ventricle and the proximal part of the aqueduct are also dilated, the latter ending in a contour resembling the knuckles of a fist. There is no dislocation of the aqueduct. The findings are probably due to a tumour in the upper part of the 4th ventricle.

operations earlier, or shortly before or after the Torkildsen procedure, and in these cases it is difficult to decide the significance of the Torkildsen procedure with regard to improvements obtained and the risk of operation. They have therefore been excluded, and the present report comprises 22 cases. The hydrocephalus was due to Sylvian aqueductal obstruction in 4 cases, and to tumours in 18. In 2 of the cases of aqueductal stenosis an unsuccessful attempt was made to relieve the obstruction by forcing through a rubber catheter.

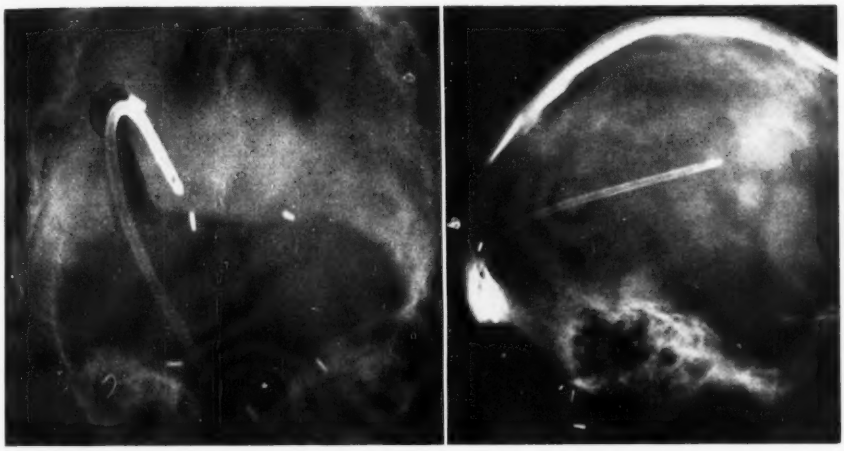
The mortality following operation is high in this material. Within 1 month 9 patients died. It must be taken into consideration, however, that several of our patients had been admitted to the hospital in an advanced stage of their disease. Of the 9 patients who died, 5 were so ill on their arrival at the hospital that operation was performed as a last resort.

Six patients died later. One (No. 8) who had an unverified tumour centrally localized to the posterior part of the 3rd ventricle, was much improved on discharge from hospital, but died 1 month later. The cause of death is not known.

Of 2 patients with an unverified tumour in the 3rd ventricle, 1 (No. 2) lived for 11 months after operation, and 1 (No. 11) for 5 months. Both were definitely improved for the greater part of this time.

One patient (No. 9), with a malignant tumour in the brain stem, lived for 6 months after operation, but no improvement was observed postoperatively.

Another patient (No. 3), with Sylvian aqueductal stenosis, lived for



FIGS. 2 and 3. Case 6. Roentgen control of the tube after operation. Note occipital bone defect after exploration of posterior fossa.

almost 5 years after operation, but there was no improvement. An infection occurred in the postoperative period which had in all probability destroyed the communication for the CSF with the cisterna magna.

Finally, a 9-year-old boy (No. 4), with an unverified tumour in the region of the 3rd ventricle, survived the operation for 9 years and, apart from poor vision, enjoyed good health during this time. In this case it must be considered that the operation had the desired effect, and that death was due to a gradual growth of the tumour.

Among the 7 patients who are still alive, 1 (No. 6), a woman aged 22 at the time of operation in 1939, remains completely free from all signs and symptoms of disease (Figs. 1, 2, 3). Ventriculography in this case suggested a tumour in the upper region of the 4th ventricle, blocking the caudal orifice of the aqueduct. The posterior fossa was explored and the findings of the ventriculogram confirmed. It was impossible to remove the tumour, which was attached by a broad base to the floor of the ventricle, and no biopsy was performed.

Of the remaining patients, 3 were operated upon in 1947, and 3 in 1948. In all 6 there has been considerable improvement since operation. Their

FIG. 4.

FIG. 5.

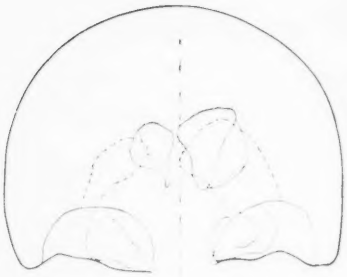


FIG. 6.

FIG. 7.

FIGS. 4–7. Case 13. Lateral and frontal views of ventriculograms, with diagrams according to Lysholm.[6] No obvious lateral displacement. The left lateral ventricle is somewhat widened. The right anterior horn is slightly flattened on the right side and pushed back about 1 cm. The floors of both anterior horns are elevated, slightly more on the right. The septum pellucidum is thickened and tilted to the right. The 3rd ventricle is completely compressed near the foramen of Monro. The right temporal horn is flattened on the top and in front.

general condition is excellent. In 4 of them (Nos. 14, 15, 20, 22) vision is considerably reduced, but it was already poor before operation. In the other 2 (Nos. 12, 13), both of whom had tumours, there has been a remarkable improvement in vision in addition to the general improvement. In Case 13

postoperatively there was high intracranial pressure, with choked discs of intense degree, diminishing vision, and disorientation (Figs. 4, 5, 6, 7). A dye test showed no CSF circulation. At the time of transfer to his local hospital for postoperative irradiation his condition was not at all satisfactory. Later on, however, he improved to such an extent that, according to his own view, he was completely restored to health, even as regards his vision. This improvement is probably due to a spontaneous re-establishment of CSF circulation; it is rather difficult to attribute it entirely to the radiation treatment. Case 12 is similar to Case 13, although in this instance the dye test showed circulation of the CSF.

A review of the 22 cases is given in chronological order in Table 1. Vision is given after correction of errors of refraction, if existing.

DISCUSSION

According to experiences reported from different clinics, the Torkildsen procedure has proved to be a valuable palliative operation in certain cases of hydrocephalus due to obstruction within the ventricular system, resulting in the blocking of the CSF circulation. In this clinic the Torkildsen palliative operation has been used particularly in cases of tumour in the region of the 3rd ventricle. In cases where a radical operation seems possible an attempt is always made to remove the tumour in the first place. The indications for ventriculocisternostomy have thus been more restricted than in the Torkildsen series. In cases of atresia of the aqueduct of Sylvius re-establishment of the natural communication has, as a rule, been aimed at, and consequently Sylvian aqueductal obstructions are scarce in this material (Nos. 3, 7, 10, 20). Since 1944, at the proposal of Leksell,[5] the majority of Sylvian aqueductal obstructions have been operated upon by probing the aqueduct and reconstructing its lumen by inserting a permanent spiral of thin wire, usually tantalum. The results hitherto have been encouraging.

Torkildsen's experience that even if intracranial pressure remains at a high level for a considerable time after operation, the ventriculocisternostomy can function satisfactorily, is confirmed in this material (Nos. 12, 13). It is not always easy to say whether a temporary obstruction of the tube is the cause, as one is not inclined to perform dye tests if the patient soon after operation is in poor condition and has increased intracranial pressure. In Case 13 the result of the dye test was negative, although a passage seems to have been established later on spontaneously. According to the author's experience it may be necessary to draw off 30–40 cc. of CSF by lumbar puncture on adult patients 45-60 minutes after injection of dye into the ventricular system before one can be sure whether communication is present or not.

As shunting tubes ordinary rubber catheters of varying size have been used, not differing essentially from the practice in other clinics. We have found that rubber is a useful material, as previously emphasized by Torkildsen[11] and Brown.[1] From these experiences it is not, of course, possible to

TABLE 1

Summary of 22 cases of ventriculocisternostomy (VC)

Case Sex, Age	Duration Illness	Dominant Symptoms	Ophthalmic Status Before Operation	Ventriculography	Operations	Postop. Course	Ophthalmic Status on Discharge	Communication Test	Roentgenotherapy	Path. Anat.	Results
1. B.B. M, 52	8 mos.	Drowsiness, poor memory, unsteady gait, failing vision. Condition poor.	Vision not tested. Choked discs: R 1D, L 1D.	Hydroceph. Pontine lesion.	Feb. 1939: Explor. post. fossa. VC.	Died after 5 days.			−	Glioma corpora quad.	
2. I.K.J. F, 25	2½ yrs.	Headaches, vomiting, diplopia, failing vision. Left hemianopsia.	Vision: R light percep., L 0.7. Choked disc: L 1D. Atrophy: R.	3rd vent. lesion.	May 1939: VC.	Uneventful.	Vision: R & L light percep. Atrophy: R & L.		+		Poor vision. Otherwise well. Died 11 mos. postop.
3. L.I.S. F, 10	3 yrs.	Headaches, vomiting, failing vision.	Vision: R 0.7, L 0.1. Atrophy: R & L.	Symmetrical hydroceph. Stenosis Sylv. aqued.	Aug. 1939: VC. Reop. for CSF cyst 11 days later.	CSF cyst. Meningitis.	Vision: impossible to estimate. Atrophy: R & L.		−		Not improved. Died after 5 yrs. (attributed to reop.).
4. S.R.A. M, 9	2 mos.	Headaches, vomiting, diplopia, failing vision.	Vision: R 0.4, L 0.4. Choked discs: R 4–5, L 3–4 D.	Lesion near 3rd vent. Blocked F. Monro.	Sept. 1939: VC. Reop. for CSF cyst 27 days later.	CSF cyst.	Vision: R 0.1, L 5/80. Atrophy: R & L.	Passage of dye.	+		Poor vision. Lived in good condition for 9 yrs.
5. K.J.P. M, 7	2 mos.	Nausea, vomiting, unsteady gait, weight loss.	Vision: R 0.8, L 0.5–0.6. Choked discs: R 5–6, L 6D.	Symmetrical hydroceph. Sylv. aqued. elevated, curved, compressed. Pontine lesion.	Sept. 1939: Explor. post. fossa. Arch of atlas removed. VC.	Sudden death 2 days postop. from pressure on medulla.			−	Medulloblastoma pons.	
6. E.J.A. F, 22	2 mos.	Headaches, vomiting, diplopia, failing vision.	Vision: R 0.9, L 0.9. Choked discs: R 4, L 5D.	Blocked Sylv. aqued. Probably tumour in upper region 4th vent.	Oct. 1939: Explor. post. fossa. Tumour identified. No biopsy. Arch of atlas removed. VC. Reop. for CSF cyst 3 wks. later.	CSF cyst.	Vision R 0.9, L 0.9. Choked discs: R 2, L 1–2D.		−		Living & well 10 yrs. postop. Last report July 1949.
7. I.E. F, 5	2½ yrs.	Epileptic fits. Mentally retarded. Unsteady gait. Enlarged head.	Vision not tested. Choked disc: L 1D, R pale.	Symmetrical hydroceph. Stenosis Sylv. aqued.	Aug. 1941: Explor. post. fossa. Probing of aqued. attempted. VC.	Died after 6 days.			−	Aqued. obliterated ½ cm. Congenital atresia.	
8. S.H.L. F, 36	5 yrs.	Headaches, vomiting, failing vision. Diagnosed 1939. Improved after radiation. Symptoms recurred 1943.	Vision: R & L light percep. Atrophy: R & L.	Central lesion, bulging into post. part 3rd vent., compressing Sylv. aqued.	Sept. 1943: VC.	Uneventful.	Status quo.	Passage of dye.	−		Died 9 wks. postop., cause unknown.

after radiation. Symptoms recurred 1943.	Atrophy: R & L.	3rd vent., compressing Sylv. aqued.						op.			op., cause unknown.
9. I.V.S. M, 13	1½ yrs.	Polydipsia, polyuria, anorexia, vomiting, failing vision, headaches.	Vision: R & L light percep. Atrophy: R & L.	Stenosis Sylv. aqued. on leaving 3rd vent., probably due to tumour.	April 1946: VC.	Uneventful.	Status quo.	No passage of dye.	−	Malignant tumour, possibly papilloma.	Not improved. Died 6 mos. post-op.
10. K.A.G. M, 19	5 yrs.	Headaches, numbness left cheek, failing vision. Diagnosed 1942. Good condition & op. not advised. Symptoms recurred 1946.	Vision: R 0.8, L 0.4. Discs: No protrusion.	Symmetrical hydroceph. Stenosis Sylv. aqued.	June 1946: VC.	Meningitis. Died after 7 days.			−	Stenosis Sylv. aqued. probably congenital.	
11. J.A.B. F, 40	10 mos.	Headaches, vomiting, stiff neck. Right hemiparesis after attack of unconsciousness. Nystagmus. Hypertension	Vision: R amaurosis. L 0.8. Choked discs: R 3, L 4D.	3rd vent. lesion.	March 1947: VC.	Uneventful.	Status quo.	Passage of dye.	+		Improved & symptom free. After 5 mos. rapid deterioration & death within few wks.
12. S.A.O. M, 15	6 mos.	Headaches, vomiting, fatigue. Slight left hemiparesis.	Vision: R 1, L 1. Choked discs: R 2–3, L 2D.	Central lesion. 3rd vent. compressed & pushed to left.	April 1947: VC. Reops. for CSF cysts 3 & 9 wks. later.	High intracran. press. Vents. drained. Penicillin given.	Vision: Impossible to estimate. Choked discs: R 4, L 3D.	Passage of dye.	+		Improved. Works as gardener. Reads without difficulty. Last report Nov. 1949.
13. S.E.K. M, 26	1 yr.	Headaches, vomiting, dizziness, fatigue, failing vision.	Vision: R 1, L 0.6. Choked discs: R 4, L 5D.	Central lesion extending frontally & into sept. pellue.	Oct. 1947: VC.	High intracran. press. Condition poor. Disorientation & spasticity.	Vision: R 0.2, L 1/50. Choked discs: R 5, L 7D.	No passage of dye.	+		Improved. Works regularly. Says vision normal. Last report June 1949.
14. S.E.O. M, 33	5 mos.	Rapidly failing vision, headaches, dizziness, fatigue.	Vision: R 0.1–0.2, L 1/50. Choked discs: R 5, L 6D.	Large central lesion growing into both lat. vents. & 3rd vent.	Nov. 1947: VC.	Uneventful.	Vision: R 0.5/50, L 1/50. Choked discs: R 4, L 4D.	No passage of dye.	+		Improved. Gen. condition good. Vision improved since discharge. Last report Aug. 1949.
15. A.S.K. M, 22	3 mos.	Headaches, vomiting, failing vision.	Vision: R amaurosis, L counting fingers at 30 cm. Choked discs: R & L 9–10D.	Lesion in & near 3rd vent.	Jan. 1948: VC.	Uneventful.	Vision: Status quo. Choked discs: R 5, L 6D.	Passage of dye.	+		Improved. Gen. condition good. Vision unchanged. Last report June 1949.
16. S.M.K. M, 28	2 mos.	Headaches, stiff neck, dizziness, unsteady gait. Valvular heart disease.	Vision: R 0.6, L 0.5. Choked discs: R 5, L 7D.	Central lesion, bulging into right lat. vent.	April 1948: VC.	High intracran. press. Fever. Disorientation. Died after 6 days.			−	Astrocytoma.	

Case Sex, Age	Duration Illness	Dominant Symptoms	Ophthalmic Status Before Operation	Ventriculography	Operations	Postop. Course	Ophthalmic Status on Discharge	Communication Test	Roentgenotherapy	Path. Anat.	Results
17. K.P.L. M, 50	2 yrs.	Diplopia, headaches, dizziness, unsteady gait, disorientation, nystagmus. Poor condition.	Vision not tested. Choked discs: R & L 4–5D.	Central & subfrontal lesion.	June 1948: Bilateral VC.	Fever, disorientation, stiff neck. Died after 13 days.		No passage between lat. vents. but dye to lumbar sac.	–	Glioma.	
18. P.E.A. M, 15	3 mos.	Headaches, vomiting, diplopia, dizziness, failing vision. Poor condition.	Vision not tested. Choked discs: R 7, L 11–12D.	Lesion in pineal region.	July 1948: VC.	High intracran. press. Died after 2 days.				Tumour in pineal region. Not classified.	
19. G.I.L. M, 21	1 yr.	Headaches, vomiting, failing vision. Semicomatose condition.	Not tested.	Central lesion growing in sep. pellue., thalamus & bulging into roof 3rd vent.	Aug. 1948: VC. Reop. 6 days later for obstruction tube. Reop. 2 wks. later for CSF cyst.	Wound infected. Died 28 days after 1st op.	Choked discs: R 5, L 8D.	No passage of dye.	–	No necropsy.	
20. M.W.J. M, 17	1 yr.	Headaches, vomiting, failing vision.	Vision: R 0.5/50, L 0.3. Pale choked discs: R 3, L 6D.	Symmetrical hydroceph. Stenosis Sylv. aqued.	Oct. 1948: Explor. post. fossa. Probing of aqued. attempted. VC. 2 reops. for CSF cysts.	Gradually diminishing vision.	Vision: R amaurosis, L 1/60. Atrophy: R & L. Choked discs: R 1, L 3D.		–		Improved. Gen. condition good. Vision improved, can read headlines. Last report Aug. 1949.
21. H.A.M. M, 33	6 mos.	Headaches, attacks of dizziness with numbness tongue & upper lip. Failing vision, increasing drowsiness. Condition poor.	Vision: R 0.1, L 0.2. Choked discs: R 11, L 9D.	Lesion bulging into middle & post. part right lat. vent. & ant. part 3rd vent.	Oct. 1948: VC.	Sudden death after acute hyperthermia 7 days postop.			–	Meningioma choroid plexus. Malignant degen.	
22. L.A.K. M, 14	5 yrs.	Headaches, vomiting, diplopia, failing vision. Diagnosed 1943. Spontaneous improvement; op. not advised. Deterioration June 1948.	Vision: R & L amaurosis. Choked discs: R & L 8–9D.	Symmetrical hydroceph. Impossible to fill 3rd vent. with air.	Oct. 1948: VC.	Uneventful.	Vision: R & L amaurosis. Choked discs: R & L 5D.		+		Improved. Gen. condition good. Almost blind. Last report Aug 1949.

conclude that rubber is the best material or the only conceivable one. Penfield's[8] observation that the tube does not change its form or position on movement of the cervical spinal column suggests the possibility of using a stiff tantalum tube soft enough to be moulded at operation. Plastic tubes might also be considered in this connection.

In the great majority of cases of tumour reported here radiation treatment has been given postoperatively. In such cases it is, of course, impossible to decide whether the operation or the radiation treatment has been the main factor in any improvement that may have occurred. If the circulation of CSF has been blocked by a centrally growing tumour, however, it does not seem probable that roentgenotherapy alone will be able to re-establish the passage of CSF. Case 6 is still completely free from symptoms 10 years after operation, and was not given radiation treatment.

In a few cases the posterior fossa has been explored during operation in connection with the Torkildsen method (Figs. 2, 3). In this material there is no indication that exploration of the posterior fossa increases the disposition to postoperative complications, such as CSF cysts.

Several of the patients in this material were in a poor condition on arrival at hospital, and most of them could not have been operated upon by more radical methods. The opinion that the Torkildsen procedure is a valuable palliative operation has therefore been confirmed at this clinic.

SUMMARY

The possibilities of surgical treatment for relief of hydrocephalus caused by blocked circulation of CSF within the ventricular system are discussed. A survey of 22 patients operated upon according to the Torkildsen procedure is given.

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FAILURE TO DEMONSTRATE GLIOMAS WITH EVANS BLUE

PRECAUTIONS IN USE OF INTRAVITAL DYES IN MAN

WILLIAM H. SWEET, M.D.*

Neurosurgical Service, Queen Elizabeth Hospital, Birmingham, England

(Received for publication January 21, 1950)

ABOUT 8 years ago I injected intravenously the azo dye Evans blue, T 1824, into 2 patients with intracranial tumours in the hope that it might concentrate differentially in the neoplastic tissue and enable the surgeon to make a sharper distinction between normal white matter and white matter invaded by tumour. Evans blue was chosen since Duran-Reynals[2] had found in normal and tumour-bearing animals that healthy sarcomas and carcinomas localized the dye, whereas necrotic tumour and the normal central nervous system were the only tissues impervious to it. A large experience had already accumulated in man demonstrating its harmlessness and Brunschwig, Schmitz and Clarke[1] had found visible discolourations in and about malignant neoplasms in 20 of 30 human patients after 30–100 mg. of this dye were injected intravenously.

The dye proved to be unsatisfactory not only because it was not apparent to the naked eye in gliomatous tissues, but also because it stained the patient's skin undesirably and for a protracted interval in the amounts used. The results were reported at a meeting of the Society of British Neurological Surgeons in 1942; in view of the renewed interest in the differentiation of cerebral tumours by dyes it seems desirable to place these fruitless results on record to discourage repetition of the same tactic.

CASE REPORTS

Case 1. In D.P., a 33-year-old female, 100 mg. of the dye were injected intravenously in sterile normal saline on Mar. 12, 1942. Over the course of several hours a pronounced blue colour appeared in the skin everywhere and persisted. On March 15, another 50 mg. of the same dye were injected intravenously; a pimple on the skin was noted to be a much deeper blue than the remainder of the skin. On Mar. 16, 1942 osteoplastic craniotomy disclosed a left frontal meningioma which was intensely blue, whereas the colour of the brain, including that part adjoining the tumour, was normal. The encapsulated meningioma was, of course, readily distinguishable from normal brain without the aid of the dye. On microscopic examination a highly fibroblastic type of neoplasm was seen, the collagen of which was stained blue. This led to the suspicion that the patient's subcutaneous collagen had also taken up the dye, a disturbing thought which was confirmed by the fact that the blue colour to the skin faded only gradually; several months elapsed before it was no longer noticeable. For her first postoperative month she was a striking pastel shade of blue. Happily she did so well on other scores that neither she nor her husband minded the colour change.

* Massachusetts General Hospital, Boston 14, Massachusetts.

Case 2. In T.B., a male aged 40 years, 100 mg. of Evans blue were injected intravenously on Mar. 17, 1942. In a few hours his skin became about as blue as that of the previous patient. On March 18 a glioblastoma multiforme in the posterior inferior part of the right frontal lobe was partially removed. This tumour was unusually well supplied with blood vessels. Despite this fact it was not possible to be certain on inspection that the grayish-red neoplasm or the adjoining cerebral white matter had taken up any of the Evans blue injected the previous day. On his 14th postoperative day meningitis developed in which β hemolytic streptococcus was the organism, and he died on April 3, 1942, 2 days later. At postmortem study his skin retained the unattractive blue colour that had developed on the day of the injection, but careful inspection of the freshly cut brain showed no more evidence of the Evans blue in tumour or adjoining brain than was seen at operation 16 days previously.

A further review of the literature at that point disclosed that Hamperl[3] had noted persistence of a marked blue-gray colour in the skin of a woman 9 years after treatment of her presumed carcinoma with another azo dye, isamine blue. The total dose of the dye was not stated but it was given both orally and parenterally. At postmortem examination granules of dye were present in the connective tissue of the papillae of the skin as well as in collagen elsewhere. Incidentally there was after this long interval no dye in the reticulum cells of the spleen or in the Kupffer cells, sites of predilection as described by most authors in the early period after injection. Proponents of the administration of the dye had alleged that it left the body in 2 to 3 months (Bernhardt, Roosen).

This report and our own experience suggest that elimination of dye from the skin is an additional desideratum to be noted with care in further searches in this field. The initial trials of any new dye staining the skin should certainly on this account involve small doses, no matter how low the toxicity of the agent.

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PARADOXICAL IMPROVEMENT IN HEMIPLEGIA FOLLOWING CORTICAL EXCISION

KEASLEY WELCH, M.D., AND WILDER PENFIELD, M.D.
Department of Neurology and Neurosurgery, McGill University, and the Montreal Neurological Institute, Montreal, Canada

(Received for publication February 13, 1950)

IMPROVEMENT in longstanding hemiparesis has resulted from complete ablation of the previously injured motor cortex. This unexpected result followed the operative removal of epileptogenic lesions in 3 cases of focal epilepsy.

David Ferrier removed the Rolandic cortex of monkeys and proved that here resided an area of motor control. The experiment had been well conceived and its significance was clear. However, observations such as we are about to record show that, although there is abundant clinical evidence to identify the deficit of the hemiplegic patient with that seen following removal of inactivation of the motor area, additional and perhaps more subtle disorder might operate as well during the course of human disease.

Improvement in the control of hemiparetic limbs as the result of excision of sensorimotor cortex seems to constitute a clinical paradox. In each of the patients to be reported, sensorimotor convolutions were excised in order to free the patients of epileptic attacks. In each case the hemiparesis was not made worse, but was actually improved. The observations that we have made are presented in the hope that they may cast light upon the neural mechanisms involved in hemiplegia, spasticity and recovery of motor function in man.

OBSERVATIONS

Before summarizing our observations, abstracts from the records of the 3 patients will be presented.

Case 1. L.W. A 22-year-old girl, was first seen Oct. 12, 1946. She complained of weakness of the right arm and leg and of seizures which were ushered in by sensation in the right arm and movement of the arm.

The weakness of the right arm and leg was first noted when she was a baby of 1 month and epileptiform attacks made their appearance at 11 years. In an attempt to stop the seizures she was operated upon that year by Dr. Charles Frazier. He discovered in the left hemisphere a porencephalic cyst which he unroofed. Attacks became less frequent for a year, stopped for 4 years and then recurred.

Examination in Montreal showed the patient to be a tall left-handed young woman. The right arm and leg were small by comparison with the other side. Movement of these extremities was limited. Her chief difficulty in using them was the stiffness produced by spasticity. She walked with a pronounced limp and used the hand for nothing more than the simplest actions.

The tendon reflexes were exaggerated on the right side and the plantar response was extensor. Two-point discrimination and position sense were severely impaired

on that side and examination of the visual fields showed that she had a right inferior quadrantic anopsia.

EEGs gave evidence of a localized epileptogenic area in the left frontal region. "Spikes" and "sharp wave" variations in potential were recorded consistently from this area.

1st Operation. Left craniotomy and partial removal of meningocerebral cicatrix. On Oct. 16, 1946, the left hemisphere was exposed through the cranial defect left at the time of the previous operation. The scalp was found to be adherent to dura and the dura to brain. Anteriorly the frontal lobe appeared quite normal but for a distance

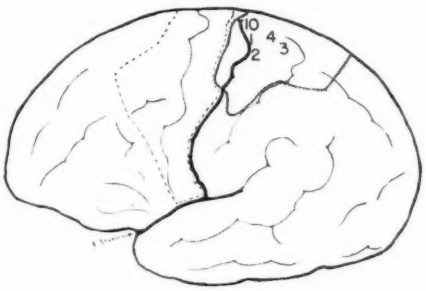

FIG. 1. *Case 1.* Interrupted line=extent of first excision. Crossed line=2nd excision. The stimulation results were obtained at the 2nd operation, utilizing the Rahm stimulator, frequency 60, 3 V. Point 1: Movement of 3rd, 4th, and 5th fingers of right hand. Point 1: (Patient was asked to keep her hand still.) Tingling of ulnar side of hand. Point 3: Sensation in right arm followed by movement of this arm. Point 4: Sudden movement of right arm and a feeling like that which habitually occurred before an attack, 1½ V., Point 10: Tingling in right leg.

of 3 or 4 cm. from the edge of the former opening backward there were dense adhesions.

The patient had two spontaneous seizures during the early stages of the operation. The operation was carried out under local anesthesia but stimulation produced no response from the exposed cortex, possibly because of the attacks. A large excision of abnormal cortex was made in the precentral region (Fig. 1).

Course. The attacks continued to occur, though with decreased frequency. Consequently 8 months later a second operation was undertaken.

2nd Operation. Left craniotomy and removal of meningocerebral cicatrix including the remainder of central area. The brain was exposed a second time using local analgesia. Stimulation now produced both sensory and motor responses in arm and leg as shown in Fig. 1. It was thought that this responsive area was part of the postcentral gyrus. There was some cerebral tissue anterior to this gyrus, but no response was obtained upon stimulating this. In the central portion of the hemisphere there was a large fluid-filled cavity. No other responses were obtained by stimulation of its banks.

It was decided to complete the removal of all cortex surrounding the central area of destruction. The patient's strength of voluntary movement of hand and foot was tested. She was warned that in order to stop the attacks we would have to increase the weakness of hand and foot. The responsive convolution and all other convolutions above the fissure of Sylvius which formed the posterior border of the area of destruction were then removed.

The anesthetist, Dr. Nitikman, then tested strength in the opposite hand and foot and found no decrease whatever!

Postoperative Course. Careful study showed that there was an increase in the two-point and position sense deficit but no increased paresis.

When seen 2 years after operation, she had had no postoperative convulsive

seizures. However, it was clear that there was a remarkable change in the patient's hemiparesis. Instead of carrying the paretic upper extremity in a flexed and spastic manner as she had done before, she now kept her arm extended by her side (see Fig. 2). The muscles were plastic. She was beginning to use the hand for eating which had never been possible before. In walking she could swing her leg without the former spastic stiffness so that her hemiplegic limp had actually disappeared. She had spent the year doing satisfactory university work.

FIG. 2. *Case 1.* Photograph taken 1 year after the 2nd operation. The patient was then able to hold the small right arm at her side rather than in a flexed adducted attitude.

The actual removal of tissue in the two operations was confined to cortex. In Fig. 1, the extent of removal by both operations is indicated. Some improvement in function may have followed the first operation but it was certainly accelerated by the second procedure.

Case 2. A.A., a 12-year-old girl, was first admitted to the Montreal Neurological Institute June 21, 1947, complaining of seizures.

She was well until she was 2½ years of age when she had a severe febrile illness with a temperature which is said to have reached 107° F. A generalized seizure occurred at that time and for 6 days afterward there were numerous bouts of muscular twitching. Then another severe seizure involving the left arm, face and leg occurred and following this attack the left arm and leg were paralyzed.

For 2 years preceding her admission the child had seizures characterized in onset by a flexion and elevation of the left arm, and for 1 year she had in addition small attacks in which she was unable to speak.

On *examination* it was found that the left arm and leg were smaller than those on the right. The patient walked with a spastic gait, the left arm held rigidly flexed and adducted. There was left spastic hemiplegia with hyperactive tendon reflexes and plantar extension on that side.

X-rays of the skull showed a small right hemicranium. Pneumocephalogram revealed cerebral atrophy most pronounced on the right, and lowering of the floor of the right lateral ventricle suggested that this process involved the basal nuclei as well.

EEGs revealed high voltage "sharp waves" and rhythmic disturbances at 2 to 3/sec., maximal from the right frontal region.

After a trial of medical management, she was readmitted Jan. 5, 1948, because attacks had continued.

1st Operation, Jan. 10, 1948. Right osteoplastic craniotomy and removal of atrophic area of brain. There were adhesions between brain and dura, particularly in the region of the Sylvian fissure. This fissure was wide due to underlying atrophy, and there was an area of small, leathery gyri posterior to the central sulcus and above the fissure of Sylvius. A very small gyrus, which joined both the pre- and postcentral gyri above, was the downward continuation of both.

Stimulation produced movements in the paretic extremities though at somewhat higher threshold than average (Fig. 3).

Excision included the pre- and postcentral gyri of the arm subdivision and below,

down to the fissure of Sylvius as well as the shrivelled parietal gyri (Fig. 3). When the excision was completed she could move her arm as well as before.

Course. There was never any increased motor disability postoperatively. The patient was readmitted March 10, 1948, because attacks had continued. Examination disclosed that her spasticity was less severe than before in both the arm and leg on the left side. The decrease in spasticity was most marked in the elbow, although her gait had improved as well.

2nd Operation. April 3, 1948. Right osteoplastic craniotomy and removal of atrophic area of brain. There were adhesions beneath the dura. The banks of the previous removal were satisfactory. The patient's attacks could be reproduced by stimulation

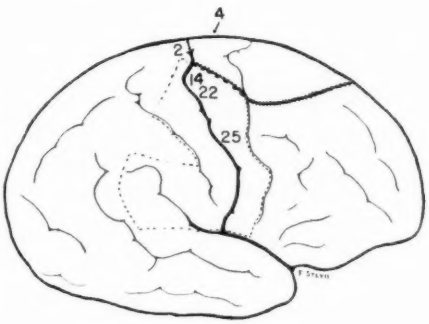

FIG. 3. *Case 2.* Interrupted line = extent of excision at 1st operation. Crossed line = 2nd excision. At the 1st procedure stimulations were made with the Rahm stimulator, frequency 60. 2½ V., Point 14: Movement at elbow. 3 V., Point 22: Pronation of left arm continuing for 10 seconds after withdrawal of stimulus. 3 V., Point 25: Slight pronation of hand with afterdischarge.

At the 2nd operation, stimulations were made with the Rahm stimulator, frequency 60, 2 V. Point 2: Sensation in left thigh. Point 4 (1 cm. within the sagittal fissure): Sensation in foot. Movement of the entire leg was obtained by stimulation in the neighborhood of point 4 but this motor point was not numbered.

anterior to the upper part of the precentral gyrus and a large removal was carried out (Fig. 3).

Postoperative Course. There was no increase in paresis in the contralateral extremities, and spasticity in both the arm and the leg was less pronounced than before.

Case 3. A.M., a 25-year old woman, was admitted to the Montreal Neurological Institute, Feb. 14, 1937, complaining of seizures for 15 years.

The patient had been born spontaneously after transverse presentation. At the

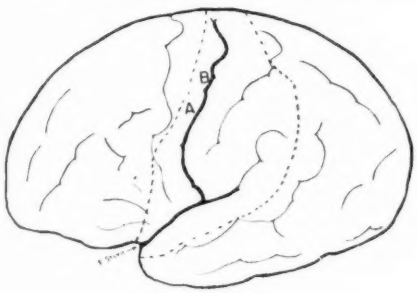

FIG. 4. *Case 3.* Interrupted line = extent of excision. The Thyratron stimulator was used, frequency 60, intensity 4. Point A: Swallowing and closure of mouth. Point B: Slight inward rotation of right arm.

age of 1 week her mother noticed that her right arm and leg were weak, and walking was delayed until the age of 3 years on this account.

Fifteen years before her admission she began to have seizures characterized, in

onset, by a pounding sensation at the top of the head followed by shaking of the right arm. These attacks became generalized.

Examination disclosed spastic hemiplegia with exaggerated deep tendon reflexes and plantar extension on the right side. There was impairment of position sense and stereognosis on the right, and smallness of the right extremities.

Operation. Feb. 18, 1937. Left osteoplastic craniotomy and removal of cerebral cicatrix. The brain was seen to have undergone atrophy in the region of the Sylvian fissure and extending upward and backward beyond the limits of this fissure. The precentral region was atrophic up to the midline.

The stimulation results identified the central sulcus. A large excision including pre- and postcentral gyri was carried out (Fig. 4.)

Postoperative Course. There was no increased weakness of the paretic extremities. There was an homonymous hemianopsia.

Summary of Results. Large removals of sensorimotor cortex were made in 3 patients who had been hemiplegic since infancy and who exhibited focal cerebral seizures. No increased paresis was seen in the contralateral extremities following operation although the excised cortex had been electrically excitable. There was decreased spasticity in the paretic extremities after operation, and as a result, motor performance was improved.

DISCUSSION

The Nature of the Deficit. Since the results of these excisions are clearly unusual* it is well to examine the pathological state of the sensorimotor areas in these cases from anatomical and functional viewpoints.

Anatomically each of these patients showed an objective lesion involving the Rolandic region and other portions of the exposed cortex as well. A large porencephalic cyst with surrounding atrophic gyri was found in Case 1. This must have been present from birth and resulted from birth injury or some ante-natal cause. The cerebral atrophy seen in Case 2 followed an infection at the age of 2 years, and the mechanism of injury was probably thrombosis of cerebral veins. In Case 3, the atrophy was due to obstruction of the middle cerebral artery.†

Each of these patients showed a functional deficit which indicated damage to both pre- and postcentral areas. In each, the cortex was, however, electrically excitable in that movements of the paretic extremities were produced by stimulation of the sensorimotor cortex. The responses were elicited at higher threshold than usual, however. Following the cortical excisions which have been outlined, no increased deficit in posture or movement could be found.

* It should be emphasized that the usual result of removal of the precentral gyrus even in hemiplegic patients is an increased paralysis. The cortical excisions reported here were done with the full expectation that this would occur, but with the hope that this would be less of a handicap than the seizures. In the present state of knowledge it is not possible to predict in advance which patients will show no increased deficit following such an excision and we do not advocate this approach for the treatment of spasticity.

† This case was reported, together with several similar ones, by Evans and McEachern.[4] Patient No. 2266 in their report is the patient under discussion here.

It can be concluded, therefore, that the activity of these atrophic sensorimotor areas was not essential for the performance of movements, impaired as they were in these patients. That the Rolandic cortex had retained a capacity to influence anterior horn cells is demonstrated by the responses to electrical stimulation. This suggests that the clinical deficit was partly due to isolation or de-afferentiation of the damaged but electrically excitable cortex or, in other words, that the paralysis was, in part, of supracortical origin.

Reorganization of Function. The recovery of function that occurs after injury to the motor area has been the subject of considerable interest, especially during the past 15 years. As yet, however, the factors and mechanisms involved in recovery are not clear, the experimental data from laboratory animals are conflicting, and criteria for acceptance of recovery as reorganization of function are not apparent.

Few facts pertaining to the portion of the nervous system responsible for recovery have stood the test of repeated experiment. The data that lent strong support to the idea that the ipsilateral motor area takes over motor function in monkeys[2,5] have not been entirely substantiated.[3,8,10] Kennard[6] has adduced evidence in monkeys for the importance of the postcentral and frontal association areas (on the side of the motor area lesion) in recovery, but it is questionable that this represents true reorganization in the sense that a new or vicarious function has been assumed by these cortical areas. The evidence for the participation of subcortical structures in recovery of function is entirely based upon exclusion of participation of other parts.[8]

The present observations lend no support to the concept of participation of the postcentral or intermediate precentral region in motor recovery in man as our excisions did not increase paresis. In each of the patients, however, there was evidence that the original lesion involved the postcentral gyrus as there were impaired stereognosis, two-point discrimination and position sense as well as smallness of the contralateral extremities before operation was carried out.

If true reorganization of function occurs, the sites where motor function becomes secondarily localized must depend upon the nature and extent of the original injury. The gross forces of birth injury, obstruction of a middle cerebral artery and thrombosis of cerebral veins which operated in our cases did not respect the boundaries of cortical areas and certainly involved subcortical structures as well as cortex. It is not surprising, therefore, that our observations are not in accord with those obtained in animal experimentation where the extent of the original lesion can be accurately determined and where the remainder of the brain is uninjured.

Spasticity. Spasticity has not been increased in these patients following operation. Indeed in 2 the change has been in the opposite direction and has allowed improved motor performance. Browder[1] reported decreased spasticity and consequent improvement in motor ability following large removals of parietal cortex in patients showing spastic hemiplegia. It is, of course,

impossible to attribute the decreased spasticity that we have observed to any one part of the ablation—precentral or postcentral. In any event, Browder's observations and ours show an important species difference between man and other primates. In monkeys and chimpanzees existing spasticity is increased by postcentral excision and a flaccid paralysis can be converted into a spastic one by removing the postcentral gyrus.[9]

Evidence from many sources, summarized by Magoun and Rhines,[7] indicates that, in addition to being a release phenomenon depending upon the removal of inhibitory influences, spasticity depends upon intact and functioning facilitatory systems. In our cases the damaged sensorimotor cortex seems to have reinforced the spinal stretch reflexes so that when its influence was removed depression rather than release resulted.

SUMMARY AND CONCLUSIONS

1. Injured cortex, capable of producing movement in paretic limbs when stimulated and of causing movement during epileptic discharge, has been removed without producing increase of functional disability in the extremities.

2. Excision of these areas has resulted in decreased spasticity and consequently produced paradoxical improvement in the use of the limbs.

3. The postcentral gyrus and the intermediate precentral area did not take part in reorganization of function after the original injury in these cases.

4. Sensorimotor cortex which retains the capacity to influence spinal motor mechanisms, but which plays no essential role in the voluntary control of these mechanisms, seems to have exerted a specific augmenting influence upon the spinal reflexes of the involved parts. Decrease of spasticity and increase of normal function resulted from ablation of abnormal sensorimotor gyri.

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ACUTE DEGENERATIVE CHANGES IN ADENOMAS OF THE PITUITARY BODY—WITH SPECIAL REFERENCE TO PITUITARY APOPLEXY

MILTON BROUGHAM, M.D., A. PRICE HEUSNER, M.D., AND RAYMOND D. ADAMS, M.D.

Neurological Unit and Neurosurgical Service, Boston City Hospital, and Department of Neurology, Harvard Medical School, Boston, Massachusetts

(Received for publication February 13, 1950)

DEGENERATIVE changes in pituitary adenomas and their attendant clinical manifestations occur not infrequently yet they have been mentioned in only a few of the more complete descriptions of these tumors. These changes usually consist of small hemorrhages and areas of necrosis with "cyst" formation, reparative fibrosis and sometimes calcification. Some writers relate such lesions to the apparent arrest of acromegaly or to the rapid development of dyspituitarism. However, in most instances these degenerative changes are regarded as unimportant findings without precise relationship to symptoms and without significant effect on the growth of the adenoma.

In the routine examination of the brains of individuals who have died unexpectedly we have several times discovered tumors of the pituitary gland in which widespread necrosis and/or extensive hemorrhage had occurred. Clinically, these changes were associated with a sudden onset of neurological symptoms such as ophthalmoplegia, blindness, stupor or coma and they have resulted in death within a few hours to days. Extensive lesions of this type have seldom been described. In fact, a review of the literature disclosed only 5 cases showing similar symptomatology and pathological findings. The following cases are presented, therefore, in order to call attention to these severe retrogressive pituitary lesions.

REPORT OF CASES

Case 1. (B.C.H. 115140, A 44–421) This patient, a 65-year-old negro, was hospitalized because of epigastric pains of 7 weeks' duration. Investigation revealed nothing of importance except a low blood pressure (100/70) and a slight reduction of blood chlorides. A diagnosis of adrenal insufficiency was made and treatment with sodium chloride and desoxycorticosterone was instituted.

On the night of the 16th hospital-day the patient complained of severe frontal headache, and the next morning he was found semicomatose. Temperature was 105°F. and the neck was very stiff. The right pupil measured 6 mm., the left 4 mm. and neither reacted to light or on convergence; there was a complete right ophthalmoplegia. No other abnormal signs were elicited. CSF was xanthochromic and under a pressure of 180 mm.; it contained 155 RBC, 55 neutrophilic leucocytes and 295 lymphocytes/c.mm.; total protein 182 mg., and sugar 55 mg./100 cc.

On the 21st hospital-day there was definite improvement in that the patient was able to respond to simple questions. However, he remained confused and the

right ophthalmoplegia persisted. A second lumbar puncture, 1 week after the first, revealed xanthochromic fluid under a pressure of 105 mm. of water with 20 RBC and no WBC/c.mm.

Examination. Two weeks after the onset of headache the patient was still confused and disoriented. There was ptosis of the right eyelid and only very slight movement of the right eye. Movements of the left eye were probably normal though poor cooperation interfered with accurate testing. The right pupil was dilated and fixed to light (both directly and consensually) and on convergence; the left pupil now reacted normally. Vision was greatly reduced in the right eye and slightly impaired in the left but no accurate records of visual functions were obtained. Other cranial nerves were normal and there was no weakness, reflex change or sensory deficit of the limbs.

FIG. 1. *Case 1.* View of base of skull. The dark, rounded mass of the pituitary adenoma can be seen protruding from the sella turcica.

Course. The patient's condition remained poor and he died during the night of the 29th hospital-day.

Pathological Findings. A rounded mass of dark red tissue protruded from the sella turcica (Fig. 1). On cut surface it was dark red, very soft and without recognizable structure. The pituitary gland could not be identified as such. The mass had enlarged the sella to about thrice normal size and had elevated all of the clinoid processes. Lateral expansions of the mass had compressed both cavernous sinuses. The diaphragm of the sella had been destroyed and the tumor extended above the sella for a distance of 2.0 cm. Upon removal the mass was found to be spherical with a diameter of 4.0 cm.

The optic chiasm and hypothalamus had been displaced superiorly. The chiasm and right optic nerve were flattened and the latter was grooved by the overlying anterior cerebral artery. The optic tracts were not reduced in size. There were no other gross abnormalities of the brain.

Microscopic sections showed that the intrasellar tumor had undergone almost complete necrosis. In phloxine-methylene blue preparations only the faint outlines of some tumor-cells, stained a pale pink, could be seen (Fig. 2). The majority of these cells had disintegrated and most of the tissue had a homogeneous structureless appearance. Just inside the fibrous capsule of the mass, small compressed islands of recognizable pituitary cells remained; some of these cells were necrotic. The capsule and peripheral parts of the necrotic mass were impregnated with fibrin and infiltrated by neutrophilic leucocytes. In the capsule there were large numbers of proliferating fibroblasts, neutrophilic leucocytes, lymphocytes, plasma cells and macro-

phages. The capillaries and veins were unusually prominent owing to proliferation of adventitial and endothelial cells.

In the leptomeninges around the optic nerves and chiasm there were large numbers of lymphocytes, histiocytes, a few plasma cells and neutrophilic leucocytes. Similar cells were found in some of the perivascular spaces within the chiasm. Axis cylinders of nerve fibers within the chiasm and right optic nerve were reduced in number; of the remaining fibers, some were fragmented and others ended in bulbous enlargements. Within the chiasm and right optic nerve there were many hypertrophied astrocytes and scattered microglial cells of pleomorphic forms containing small droplets of fat. Many of the nerve cells of the tuberal nuclei and mammillary

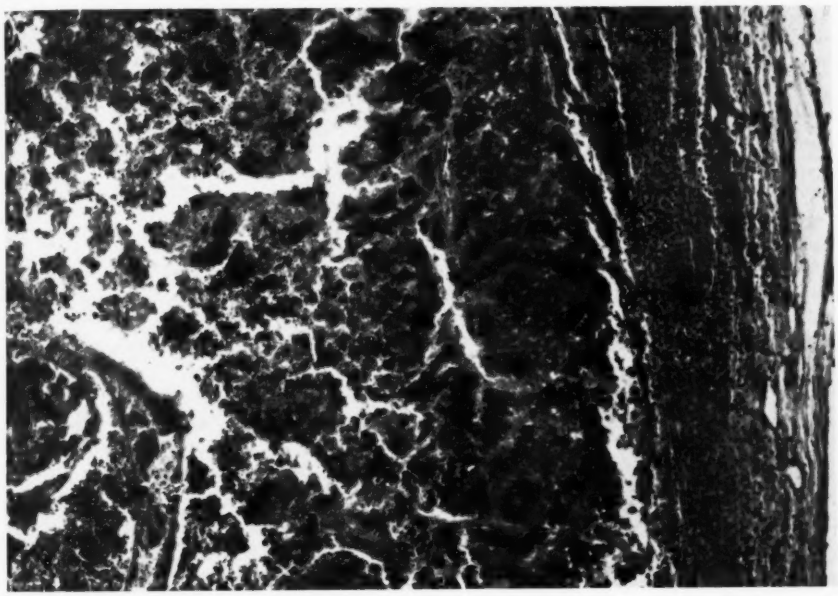

FIG. 2. *Case 1.* Phloxine-methylene blue stain (×80) of capsule of the tumor with its heavy infiltrations of neutrophilic leucocytes and lymphocytes, and of outer portion of the tumor, which has undergone necrosis. The indefinite outlines of the tumor cells are still visible.

bodies had disappeared and numerous microglial phagocytes and plump astrocytes were present. The nerve cells of the right oculomotor nucleus were swollen, chromatolytic and had eccentric nuclei but the cells of other cranial nerve nuclei were normal. Throughout the cerebral cortex there was a very slight increase in the number and size of astrocytes.

Other important pathological findings were: a well differentiated adenocarcinoma of the colon which had not metastasized; congenital absence of the right adrenal and hypertrophy of the left one; pericholangitis with focal necrosis of the liver; and focal healed pyelonephritis. The thyroid and parathyroid glands and the testes were not examined.

Final Diagnoses: (1) Adenoma of pituitary body (? chromophobe) which had undergone recent infarct necrosis; compression of optic chiasm, right II, III, IV,

and VI cranial nerves and hypothalamus. (2) Adenocarcinoma of colon (without metastases).

Comment. There was no explanation of the infarction of the pituitary gland other than that it had outgrown its blood supply. There was not enough tumor-tissue left to permit identification by special stains. The cellular reaction in the capsule of the gland and in the adjacent leptomeninges, and the pleocytosis in the CSF were probably reactions to necrosis of tissue. The compression of the optic chiasm and the right optic nerve was a result of acute swelling of the tumor. The right optic nerve had been pushed against the overlying anterior cerebral artery. The ophthalmoplegia was due to compression of ocular nerves in the cavernous sinus. The stupor and coma may have been related to acute compression of the hypothalamus but alternative explanations include an impaired cerebral circulation, and some metabolic disorder associated with severe Addison's disease. The significance of the diffuse changes in the cerebral cortex is not known but they may be structural manifestations of a metabolic disturbance of brain function. The changes in the right oculomotor nucleus were secondary to damage of the allied nerve, that is to say, an axonal reaction.

Case 2. (NP 45–33) This patient, a 46-year-old man, died on the way to the hospital. The only available history was that for several days he had felt sick and had complained of severe headaches, and that a few hours before death he had become lethargic, stuporous and then comatose. The referring physician had detected no abnormality other than drowsiness.

Pathological Findings. Examination was limited to cranial structures. When the frontal lobes were elevated a considerable quantity of blood was found. It seemed to arise from the sella which was filled with dark hemorrhagic material, and it covered the floor of the 3rd ventricle, the optic nerves, chiasm and tracts, and the olfactory bulbs. A small amount of blood had also extended along the Sylvian fissures to the lateral surfaces of the cerebral hemispheres. The blood was largely confined to the subarachnoid spaces; it was of dark purplish-red color and had clotted. Just above and slightly to the right of the sella there was a soft, round, purplish-red mass of tissue about 1.5 cm. in diameter. This was connected by a thin stalk to the remains of the diaphragm of the sella. The cerebral and carotid arteries were intact and no source of the subarachnoid hemorrhage could be demonstrated.

A block of tissue including the sella turcica, cavernous sinuses and adjacent parts of the sphenoid bone was decalcified and sectioned in the coronal plane. The sella was found to be filled with necrotic cells none of which could be identified with certainty. In most places these cells were separated by red corpuscles and did not have the usual arrangement of normal cells of the pituitary gland. In a few places they were closely packed together forming broad sheets. There were a few islands of more normally arranged though necrotic pituitary cells just inside the capsule of the gland. The extrasellar mass had about the same composition as the intrasellar tissue. Structures within the cavernous sinuses appeared to be normal. In the subarachnoid spaces around the optic chiasm and floor of the 3rd ventricle there was relatively little reaction to the hemorrhage. Neutrophilic leucocytes and lymphocytes were found in numbers out of proportion to the number of red corpuscles, and a few macrophages containing red corpuscles were present. There were no significant

changes in the optic chiasm, orbital parts of the frontal lobes or hypothalamus.

Final Diagnoses: Neoplasm of pituitary body (? adenoma); extensive necrosis of tumor; intrasellar and subarachnoid hemorrhages.

Comment. At first it was thought that the subarachnoid hemorrhage was due to the rupture of a saccular aneurysm, but none was found. Moreover, the combination of intrasellar hemorrhage, extensive necrosis of the pituitary body and an attached extrasellar mass rendered this possibility untenable. Although the histology was poor owing to necrosis of tissue and to a loss of cellular detail incident to decalcification, it was quite apparent that both the intra- and suprasellar masses were neoplastic tissue. The sella was about 2.0 cm. in greatest diameter and the intrasellar portion of the tumor was only slightly larger than the extrasellar portion. Large parts of the tumor were necrotic and the hemorrhage seemed to have arisen from necrotic vessels within the neoplasm.

Case 3. (NP 46–27) This patient, a 64-year-old man, was brought to the hospital in a stuporous state after suddenly collapsing at home. He was said to have had episodic headache and mental confusion and a steadily increasing memory defect during the previous 3 years. No further history was available.

Examination. Temperature 100°F.; pulse 75; respirations 18, B.P. 100/76. The patient was stuporous and possibly aphasic. Both pupils were small and fixed to light. There was a right hemiparesis affecting the face, arm and leg. Tendon reflexes were active and about equal in the arms but they were unobtainable in the legs. Sensory functions could not be evaluated.

Laboratory Data. WBC 11,700 with a slight increase in proportion of neutrophilic leucocytes. CSF was under a pressure of 90 mm. of water and was blood-tinged with xanthochromia of the supernatant fluid.

Course. The mental state improved over a period of 2 weeks and although a considerable degree of confusion persisted, a partial aphasia, both motor and sensory, could be demonstrated. After 3 weeks' observation the patient was committed to a psychiatric hospital where, during succeeding months, there was little change in his condition; he remained apathetic, disoriented, hemiparetic and aphasic.

Five months later, while descending a stairway, he fell to the floor striking his head in the right parietal region. He was immediately comatose. The pupils were unequal and did not react to light. There was rigidity of the arms and legs, left more than right. Ankle clonus was obtained bilaterally; the right plantar reflex was extensor and the left was flexor. CSF was grossly bloody. Respirations became stertorous and irregular; temperature rose to 102°F.; signs of circulatory collapse supervened and the patient died 48 hours later without regaining consciousness.

Pathological Findings. There was hemorrhage beneath the skin and galea overlying a linear fracture in the right parietal and temporal bones. Small fresh epidural and subdural hemorrhages, not more than a few cc. in total volume, were found on the right side.

A mass of reddish, soft tissue protruded from a markedly enlarged sella turcica to compress the floor of the 3rd ventricle and extend into the left temporal lobe and lenticular nucleus (Fig. 3). At all points this mass was well demarcated, displacing but not invading brain, and it was surrounded by a thin capsule of connective tissue. The extrasellar part of the mass was of irregular shape, measuring 6.5 cm.

in greatest diameter. The tumor was soft and gray, and parts of it were hemorrhagic. The optic chiasm was displaced to the right and the left optic nerve and tract were almost completely destroyed. The left lateral ventricle was narrow but the right was about 3 times normal size. The 3rd and right lateral ventricles were displaced to the right.

Superficial contusions and small amounts of circumjacent subarachnoid and subpial hemorrhage were found on the inferior surface of the left temporal lobe.

FIG. 3. *Case 3.* Basilar view of brain. The large multilobed tumor elevates the floor of the 3rd ventricle and the optic chiasm, and extends into the temporal lobe, where the tumor is quite dark indicating hemorrhage.

The tumor was composed of small, closely packed cells which showed a slight tendency toward alveolar arrangement. These cells, 12 to 14μ in diameter, had round hyperchromatic nuclei and relatively little cytoplasm. There were no definite mitotic figures though some of the nuclei which were very dark may have been in a phase of mitosis. In some places the cells tended to be in groups demarcated by thin strands of connective tissue; elsewhere they were arranged in broad sheets. The stroma was scanty and there were only a few, thin-walled blood vessels. Large portions of the mass were necrotic; the cells though still visible were poorly stained, eosinophilic, and had shrunken nuclei (Fig. 4). In the hemorrhagic parts there were many well preserved red corpuscles and neutrophilic leucocytes. Numerous macrophages containing hemosiderin were found in one part of the mass. Mallory's trichrome stain showed no granules in the cells. Because of the general morphology of the tissue and its apparent origin from within the sella it was assumed to be a pituitary adenoma probably of chromophobe type.

In sections of the contused temporal lobe there were areas of tissue necrosis with fresh hemorrhages, migration of neutrophilic leucocytes and shrunken pyknotic nerve and glial cells.

The only other postmortem findings of note were pulmonary congestion and oedema, and a lobular consolidation of the lower lobes of the lungs. Complete examination of the endocrine glands was not performed.

Final Diagnoses: (1) Tumor of pituitary body (? chromophobe adenoma) with extension to floor of the 3rd ventricle and left temporal lobe; hemorrhages, old and recent, and necrosis of tumor. (2) Fracture of temporal and parietal bones; epidural and subdural hemorrhages; cerebral contusions. (3) Pulmonary congestion and oedema; bronchopneumonia.

Comment. The dementia was probably related to the extension of the extrasellar tumor into the base of the brain. The abrupt occurrence of stupor,

aphasia and right hemiparesis about 6 months before death may have been due to necrosis of parts of the tumor and to hemorrhage, both within the tumor and into the subarachnoid spaces. The fresh hemorrhage in the tumor may have been caused by the head-injury.

Case 4. (B.C.H. 1217174, NP 46–44) This patient, a 70-year-old man, entered the hospital for the first time in September, 1944, because of severe intermittent headaches of about 8 months' duration. He had also noted some loss of hearing and tinnitus in the left ear and had suffered one acute attack of vertigo. The only abnor-

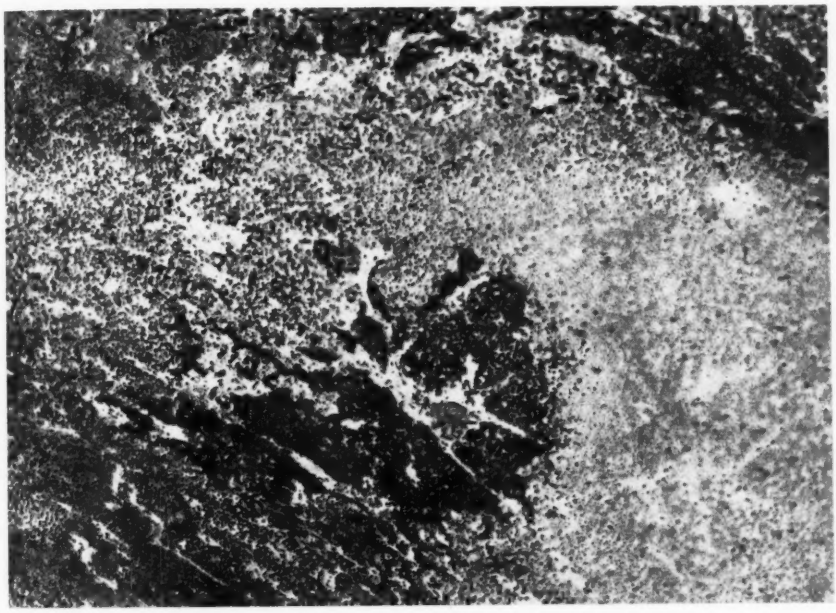

FIG. 4. *Case 3.* Hematoxylin and eosin preparation (×56) of a part of the mass in the temporal lobe showing islands of viable cells in the center and right upper corner, and extensive necrosis elsewhere.

mal signs noted at this time were inequality of the pupils (right larger than left), ptosis of right eyelid and diminution of hearing in left ear (nerve deafness). CSF pressure was 135 mm. of water; the fluid was acellular but its total protein was 78 mg./100 cc. X-rays of the skull showed marked thinning and elevation of the posterior clinoid processes and depression of the floor of the sella. The patient refused further investigation and left the hospital against the advice of his physician. His condition was said to have remained about the same until July, 1946 when, 2 days prior to re-entry, a sudden onset of severe headache was followed by nausea, vomiting and rapidly deepening stupor.

Examination. Temperature 107°F.; pulse 104; respirations 22(Cheyne-Stokes); B.P. 80/50. The patient was silent and did not respond to spoken commands. If left undisturbed he lay motionless in bed unmindful of things going on about him. He would, however, withdraw his limbs from noxious stimuli. The neck was moder-

ately stiff. The eyes were turned toward the right; pupils were constricted and did not react to light. The limbs were limp and areflexic. Plantar responses were equivocal, probably extensor in type.

Laboratory Data. WBC 18,000/c.mm. with neutrophilic leucocytosis. CSF was under a pressure of 140 mm. of water; it contained 8,000 RBC and 93 WBC/c.mm.; total protein 83 mg./100 cc.

Course. The coma deepened. Temperature ranged between 106 and 107°F. despite vigorous treatment. Death occurred 6 hours after entry.

Pathological Findings. A mass of reddish-gray tissue about 2.8 cm. in diameter filled the sella turcica, extended up through the diaphragm and compressed the optic chiasm. A small amount of freshly clotted blood covered the posterior margin of the mass and occupied the adjacent subarachnoid spaces. There were small amounts of blood in the lateral and 3rd ventricles. Except for slight atherosclerosis of the large cerebral arteries, there was no other gross abnormality of the brain or of the other organs.

On microscopic examination large portions of the intrasellar tissue were seen to be necrotic and infiltrated with neutrophilic leucocytes. In the better preserved parts the cells were very close together and without special arrangement. Specific stains revealed no granules in the cytoplasm of the viable cells. The nuclei were round and quite darkly stained; there were no mitotic figures. Remnants of normal pituitary tissue were compressed into a narrow band just inside the gland's capsule. There was hemorrhage into some portions of the necrotic mass and into the adjacent meninges. The latter were infiltrated with neutrophilic leucocytes and lymphocytes.

Final Diagnoses: Adenoma of pituitary body (? chromophobe); compression of optic chiasm; necrosis of tumor; intrasellar and subarachnoid hemorrhages.

Comment. The factors leading to coma could not be reconstructed since the clinical data were limited and the autopsy was incomplete. Necrosis of and hemorrhage into the pituitary tumor were undoubtedly important. The visual fields were never examined and there was no mention of the optic discs at the time of the last examination.

Case 5. (NP 46–61) A 39-year-old housewife was first admitted to the hospital in September, 1946, with a chief complaint of headache. She was known to have had untreated diabetes during the previous 12 years. She had borne 7 children before the age of 26 years, at which time menstruation ceased and she became sterile. During the past 7 years there had been progressive coarsening of the features, deepening of the voice and steady increase in weight amounting to 20 pounds in the 6 months prior to entry. For 5 years she had been having moderately severe, intermittent headaches usually localized to the right supraorbital region. During the last year the fluid intake had increased to as much as 10 quarts daily. Three days before entry the headache became almost continuous, extremely severe and it was accompanied by diplopia. On the day of admission the patient swooned and was brought to the hospital.

Examination. The patient was an obese, lethargic, middle-aged woman with coarse features. The nose was broad and flat, the jaw prominent, the tongue large and the lips were thick. The fingers were short and stubby. There were no other abnormalities.

Laboratory Data. Fehling's test for sugar in the urine gave an olive-green color and acetone test was 4+. Fasting blood sugar was 410 mg. and blood-CO_2 combining-power was below 10 vol./100 cc. X-rays of the skull showed enlargement of the sella turcica.

Subsequent Course. The diabetic acidosis was treated with insulin and intravenous fluids. Within a few days the diabetes was controlled and the patient was discharged from the hospital on a regular dosage of insulin.

Three weeks later she was readmitted. Relatives said that she had been fairly well until a few hours before re-entry when she suddenly experienced a sharp pain in the right side of her face and then soon became disoriented and stuporous. Upon arrival at the hospital she was deeply comatose.

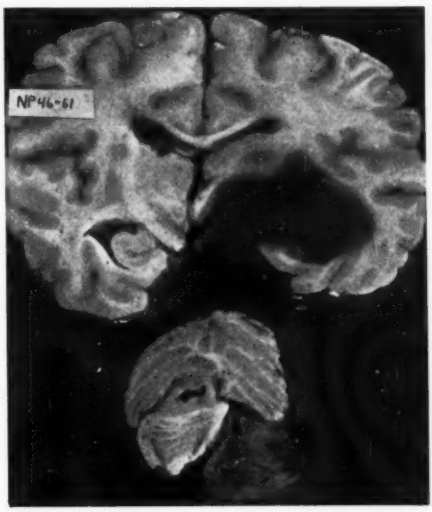

FIG. 5. *Case 5.* The large extrasellar tumor masses can be seen extending into the temporal lobe and lying on the surface of the pons. The portion of the tumor in the temporal lobe is quite hemorrhagic.

Temperature was 103°F.; pulse 80; B.P. 140/100. The right pupil measured 7 mm.; the left 3 mm.; neither reacted to light. The margins of both optic discs were blurred and there were small, round hemorrhages along the veins of both optic fundi. Plantar reflexes were extensor bilaterally. There was brick-red sugar reduction in the urine, fasting blood sugar of 345 mg. and blood-CO_2 combining-power of 39 vol./100 cc. Lumbar puncture yielded grossly bloody CSF which was xanthochromic after centrifugation. Fluids and insulin were given but the patient died 3 hours after admission.

Pathological Findings. Examination was limited to cranial structures. A large mass of soft reddish tissue protruded from the sella turcica and was connected by thin pedicles to 2 large extrasellar masses of similar appearing tissue. One of the latter projected into the medial part of the right temporal lobe; the other, whose pedicle passed through the tentorial incisura, lay in the posterior fossa. The sella was much enlarged, its clinoid processes were thin and fragile, and its diaphragm was partially destroyed. The mass projecting into the temporal lobe was oval in shape and measured 5 cm. in greatest diameter. On cut surface it was dark red, soft, rather granular, and hemorrhagic in many places (Fig. 5). It covered the optic chiasm, elevating and displacing it to the left, and extended into the brain tissue between the tuber cinereum and the medial part of the right temporal lobe. The right optic tract was severely compressed, being reduced to a thin ribbon of tissue. However, this mass was well demarcated from the brain by a thin investing capsule of connective tissue. In one place outside the capsule there was a hemorrhage which extended through the compressed brain into the temporal horn of the right lateral ventricle. The tumor in the posterior fossa was almost detached from the tumor above the tentorium. It was extremely soft, gray and measured 3×5×4.6 cm. It indented the

right anterolateral surface of the pons, the upper medulla and the middle cerebellar peduncle but it did not invade these structures. The midbrain was displaced to the left and there was a cerebellar pressure cone of moderate size.

The tumor was found to be composed of small cells about 12μ in diameter with relatively small amounts of clear cytoplasm and oval to round, rather heavily chromatinized nuclei. There was remarkable uniformity of cell type. No mitotic figures were seen. The stroma was scanty and relatively few blood vessels were found. Whereas large portions of the mass compressing the temporal lobe were necrotic, most of the mass in the posterior fossa was viable (Fig. 6). Extensive extravasation of red corpuscles was noted in many parts of the temporal lobe mass and in the circumjacent brain tissue, which was loose and fenestrated. In this region

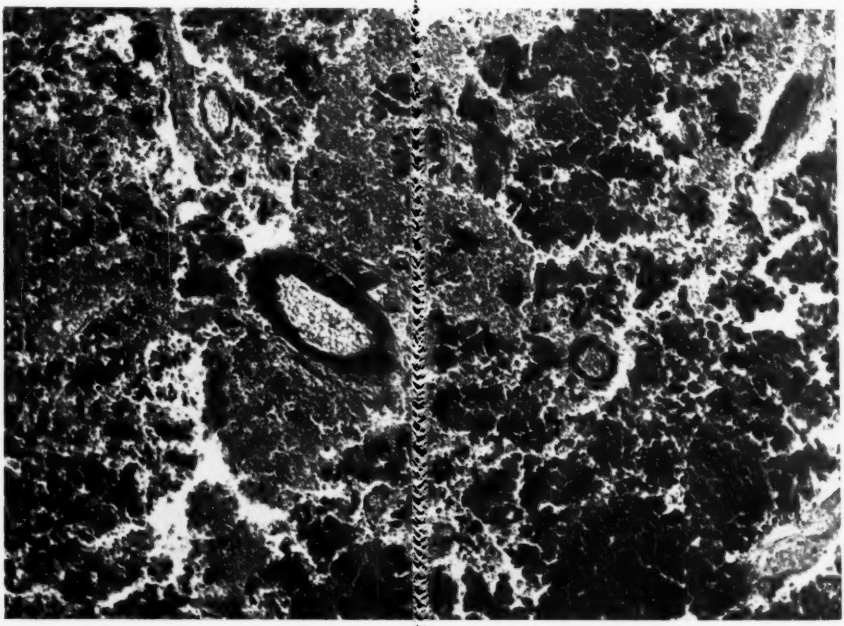

FIG. 6. *Case 5.* Hematoxylin and eosin preparation (×80) of one of the better preserved parts of the tumor, showing foci of cellular necrosis.

of the brain, nerve and glial cells were shrunken and pyknotic, and scattered neutrophilic leucocytes had migrated outside the blood vessels. The nerve cells in the mammillary bodies and those in the tuberal, ventromedial and supraoptic nuclei of the hypothalamus were compressed and darkly stained. There was no reaction to the blood in the subarachnoid spaces.

In Mallory trichrome preparations no granules could be seen but the histologic detail was poor in the parent, intrasellar portion of the tumor which had undergone extensive necrosis. Scattered cells of the anterior lobe were found just inside the capsule of the gland.

Final Diagnoses: Adenoma of pituitary body (? eosinophilic); extrasellar ex-

tensions compressing right temporal lobe, right optic tract, optic chiasm, pons and cerebellum; widespread necrosis of and hemorrhage into tumor; intraventricular hemorrhage; cerebellar pressure cone.

Comment. The diabetes mellitus, the premature menopause, the increase in weight, the deepening voice and the coarsening of features were parts of the acromegalic syndrome traceable to the adenoma of the pituitary body. The latter was presumably of eosinophilic type even though cytoplasmic granules could not be demonstrated in the viable (subtentorial) portion of the tumor. The terminal right-sided headache, coma and bloody CSF were results of hemorrhages into and necrosis of the tumor, hemorrhage into the ventricles, deformation of the midbrain, and cerebellar pressure cone.

DISCUSSION

A. *Other Cases of Hemorrhage and Necrosis in Pituitary Adenomas*

Extensive hemorrhage and necrosis in adenomas of the anterior lobe of the pituitary body are well documented pathological occurrences. There are, in all, 5 pathologically verified examples of such changes in the medical literature and at least 2 other cases in which the clinical facts are highly suggestive of the same lesions.

Bleibtreu[1] in 1905 appears to have recorded the first example of these regressive changes. In the postmortem examination of a 21-year-old acromegalic patient he discovered that the pituitary gland had been replaced by a mass of orange-colored, amorphous material representing an old hemorrhage. Clinical data were not given.

A more interesting case is that presented by Kux[10] in 1931. Here an eosinophilic adenoma had manifested itself by acromegaly and headache. While the patient was under observation there was a sudden onset of amblyopia, palsy of one of the internal rectus muscles and a drowsiness which quickly progressed to coma and, 2 days later, death. At autopsy a gross hemorrhage in a "fetal adenoma" of the pituitary gland was found. The hemorrhage had enlarged the tumor and had exerted pressure on structures in the cavernous sinuses and floor of the 3rd ventricle.

A similar case was reported by Dingley[4] who in 1932 wrote of a patient with a chromophobe adenoma of the pituitary gland in which a large, fresh hemorrhage had evidently caused a sudden collapse with coma and death within a few hours.

In 1938, Voss[13] described a 38-year-old man who had entered the hospital in a lethargic state with complete right ophthalmoplegia, a dilated, fixed left pupil, stiff neck and xanthochromic CSF. Autopsy disclosed a fresh hemorrhage into a chromophobe adenoma.

Coxon[3] in 1943 reported a case in which there was a sudden onset of severe headache followed 24 hours later by diplopia and drowsiness. Death occurred 3 weeks later and a large hemorrhage was found in an eosinophilic adenoma. In this case the CSF was clear and acellular but the total protein was 170 mg./per 100 cc.

TABLE 1

Reported cases of hemorrhagic necrosis of a pituitary adenoma

Authors	Age	Sex	Previous Symptoms	X-ray	Terminal Symptoms	CSF	Type of Adenoma	Other Pathologic Findings
1. Bleibtreu 1905	21	?	Acromegaly, 1 yr.	?	?	?	Eosinophilic	Old hemorrhage in tumor
2. Kux	?	?	Acromegaly; bitemporal hemianopia	Enlarged sella	Amblyopia; drowsiness; internal rectus palsy	?	Eosinophilic "Fetal"	Gross hemorrhage; compression floor 3rd vent. & optic chiasm
3. Dingley 1932	?	?	?	?	Sudden collapse; death	?	Chromophobe	Large fresh hemorrhage
4. Voss 1938	38	M	?	?	Semicoma; right ophthalmoplegia; dilated, fixed left pupil; stiff neck	Xanthochromic	Chromophobe	Fresh hemorrhage; compression right cavernous sinus & hypothalamus
5. Coxon 1943	?	?	?	?	Sudden severe headache; diplopia; drowsiness; 3rd nerve palsy	Clear; T.P. 170 mg.	Eosinophilic	Hemorrhage in tumor
6. Cairns 1938	?	?	Acromegaly	?	Bilateral 6th nerve palsy	?	Eosinophilic	Recovered
7. Jefferson 1940	?	?	Acromegaly	?	Sudden headache; lethargy; 3rd nerve palsy	Very bloody	Eosinophilic	Recovered
8. Brougham, Heusner & Adams *Case 1*	65	M	Symptoms of adrenal insufficiency	?	Severe headache; semicoma; stiff neck; right ophthalmoplegia; impaired vision O.D.	Xanthochromia; 155 RBC; 350 WBC; T.P. 182	?	Necrosis of entire tumor; compression cavernous sinuses, 2nd nerve, chiasm & hypothalamus
9. *Ibid.* *Case 2*	46	M	?	?	Headache; stupor; coma	?	?	Necrosis & hemorrhage in tumor; subarachnoid hemorrhage

10. *Ibid. Case 3*	64	M	Confusion; aphasia; headache; 1 episode of semi-coma	Enlarged sella	Head-injury; coma; unequal pupils; left Babinski	Bloody	? chromophobe	Old hemorrhage in tumor; skull fracture; recent subarachnoid & subdural hemorrhage; contusions brain
11. *Ibid. Case 4*	70	M	Intermittent headaches, 5 months; unequal pupils; ptosis left lid; nerve deafness A.S.	Enlarged sella	Rapid development coma; stiff neck; constricted pupils; ? bilateral Babinski	8100 RBC; 93 WBC; T.P. 83	? chromophobe	Necrosis & hemorrhage in tumor; compression optic chiasm; subarachnoid hemorrhage
12. *Ibid. Case 5*	39	F	Acromegaly; diabetes mellitus; headaches	?	Abrupt onset right facial pain; disoriented; stuporous; coma; unequal pupils	Very bloody; xanthochromic	? eosinophilic	Hemorrhage & necrosis in tumor; compression right temporal lobe, right optic tract & chiasm; ventricular hemorrhage; cerebellar pressure cone

Other single cases almost surely of the same type, but without pathological verification, are those of Cairns[2] and Jefferson.[6] In each instance a patient believed to have an eosinophilic adenoma suffered the sudden onset of an ophthalmoplegia. Jefferson's case was further marked by headache, drowsiness and sanguinous CSF. Spontaneous recovery was complete in both cases.

The cases of Van Wagenen,[12] Dott, Bailey and Cushing[5] and Sosman[11] are more difficult to evaluate because extraneous factors complicate the picture. In Van Wagenen's report of one of Cushing's patients, a head-injury just prior to death may have caused or contributed to the formation of the hemorrhage in the pituitary tumor which was demonstrated at autopsy. In Dott, Bailey and Cushing's case, 4 x-ray treatments had been given to an acromegalic woman immediately before the development of severe headache, temporal hemianopia in the left eye and stupor. Here the possibility of oedema and necrosis from roentgen therapy cannot be dismissed. Similarly, in Sosman's case a single x-ray treatment was followed within a few hours by coma due apparently to a large fresh hemorrhage into a pituitary adenoma. It is of interest that in the latter 2 cases, operative removal of the hemorrhagic and necrotic tumor resulted in prompt recovery from the neurological symptoms.

There are still other references to operative and postmortem findings of liquefaction-necrosis and hemorrhage within pituitary adenomas, as for example in the writings of Kraus[7,8,9] who regards such changes as not infrequent. In many of these cases the hemorrhages, which had the appearance of cysts filled with xanthochromic or brown fluid, were not of sufficient size to have produced neurological symptoms.

B. *The Nature of the Pathological Process*

A number of questions concerning these acute degenerative changes arise for discussion. (1) What is the essential nature of the lesions? What factors enter into their pathogenesis? (2) Are hemorrhage and necrosis separate pathological processes, or are both traceable to the same underlying circulatory disturbance, *i.e.*, hemorrhagic infarction? (3) Do these changes occur more often in one type of tumor than another? Are they expressions of the degree of malignancy of the tumor?

From our pathological material it appears that both hemorrhage and necrosis are consequences of rapid growth of the tumor. Just as the glioblastomas and the brain metastases of some carcinomas, notably those of renal cell carcinoma and chorionepithelioma, often undergo hemorrhage and infarct necrosis, so with lesser frequency do these same changes occur in the larger and more rapidly growing pituitary adenomas. It seems probable that in most of our cases the tumor had merely outgrown its blood supply. However, in our Case 1 the necrosis was so complete that a swelling of the necrotic tissue within the confines of the sella may have led to compression of blood vessels and thus to further ischemia which involved all except the

subcapsular portion of the tumor. Thrombosed blood vessels were not demonstrated in our cases, but since the tumors were not sectioned serially this process may have been overlooked. Except in Case 3 there was no evidence suggesting that trauma or other extrinsic factors had been operative.

In some cases hemorrhage and necrosis coexisted but in others they occurred separately. It will be recalled that in our Case 1 there was necrosis of nearly all of the tumor but little or no hemorrhage. In our Case 2 a widespread necrosis of both intra- and extrasellar portions of the tumor was attended by extensive hemorrhage. In our Case 3 the recent brain injury made judgment of the original lesion uncertain. In our Case 4 necrosis and hemorrhage were both pronounced while in Case 5 hemorrhage was extensive whereas necrosis was relatively slight and limited to one of the extrasellar tumors. It is therefore our impression that these 2 processes are independent.

In our material and in the cases that have been collected from the literature, hemorrhage and necrosis occurred in both eosinophilic and chromophobe adenomas (Table 1). In view of the greater incidence of chromophobe adenomas it is perhaps noteworthy that these acute changes appear to have been found more frequently in eosinophilic tumors. There are no reports of these pathological changes having been found either in basophilic, or in verified fetal-cell or transitional-cell adenomas.

The relation of acute retrogressive changes to the possible malignant character of a hypophysial growth cannot be decided conclusively. The difficulty has been in the selection of satisfactory criteria of malignancy in pituitary adenomas, a topic that has been discussed at some length by Kraus. When judged as to degree of anaplasia of tumor-cells, infiltration of adjacent structures, and metastases to distant parts of the nervous system or to other viscera, it must be concluded that all the tumors here reported were probably benign. However, in 2 of the cases the necrosis of tissue was so extensive that it was impossible to estimate the degree of anaplasia.

C. *Symptomatology*

In all of our cases and in most of those reported by others, extensive hemorrhage and/or necrosis in a pituitary adenoma was attended by a fairly uniform symptomatology. Usually a patient who has had symptoms of a pituitary adenoma for months or years develops quite abruptly headache, amblyopia, diplopia, drowsiness, confusion or coma. The CSF is under normal or slightly increased pressure, is blood-tinged, xanthochromic or colorless, and contains an increased number of white blood cells and/or an elevated protein. With the development of coma the temperature usually rises and death often ensues within a few hours to days.

There are several variations of this syndrome that warrant further exposition. As regards mode of onset and speed of evolution, it should be pointed out that whereas the symptoms and signs are usually of sudden or acute development, within hours, in occasional cases additional changes appear after an interval of some days, as in Coxon's case where a unilateral

ophthalmoplegia occurred after a delay of several days. Also, the patient may either show clinical improvement as in our Case 1, or may recover as in the cases of Cairns and Jefferson.

An altered state of consciousness, occurring in all cases except that of Cairns, was the most frequent neurological abnormality. It consisted of drowsiness, mental confusion or stupor giving way to coma within hours to days. The explanation of these symptoms is not altogether clear. They develop much too quickly to be explicable by an endocrine disorder secondary to destruction of the pituitary gland. Subarachnoid and in some cases intraventricular hemorrhage are possible causes of coma but they were not operative in our Case 1, where there was little or no hemorrhage. In this case there was histologic evidence of compression of hypothalamic structures by the swollen tumor, and such compression may have been of importance in the production of stupor. Unfortunately, the hypothalamus was not systematically examined in our other cases.

Severe headache was another frequent symptom. Details concerning the location and character of the headaches were not provided by the clinical notes on our material. Headache may be due either to an acute swelling of the tumor, or to the meningeal irritation of subarachnoid hemorrhage. Stiff neck was usually present in cases marked by subarachnoid hemorrhage but in at least 1 of our patients this sign was found in the absence of such bleeding.

Diplopia and ophthalmoplegia were frequently observed. This varied from a complete ophthalmoplegia which was usually unilateral, to the palsy of a single muscle either unilaterally or bilaterally. The oculomotor nerve was involved more often than the abducens; data concerning the action of the trochlear nerves were not available. These cranial nerve palsies were caused not by tumor infiltration, but by compression of the nerves within the cavernous sinuses. The unilateral facial pain remarked in 1 of our cases was presumably due to irritation of the trigeminal nerve.

Dimness of vision was detected in only 2 of our cases. It was unilateral in both instances, and in 1 case it was traceable to upward displacement of the optic chiasm with compression of one optic nerve against the overlying anterior cerebral artery. The only other neurological abnormality of note was a right hemiparesis with aphasia which occurred in our case with tumorous compression of and hemorrhage into the left temporal lobe.

D. *Diagnosis*

Difficulties in diagnosis are to be expected particularly in cases that first come under observation after the supervention of coma. All of the frequent causes of coma must be considered and pituitary apoplexy will suggest itself only if there is a clear history of pre-existing symptoms of a pituitary adenoma, or if there are evident physical stigmata of endocrine disease, or if there is sellar deformity by x-ray examination. In those cases of known pituitary adenoma in which the patients become drowsy, stuporous or comatose,

at least 3 other conditions incident to the growth of such tumors must be distinguished. They are: (1) An acute attack of adrenal insufficiency. (2) The development of a temporal lobe pressure cone caused by an extrasellar extension of tumor and/or hemorrhage into the compressed temporal lobe. (3) Convulsions.

It is well known that adrenal insufficiency may develop as a consequence of a pituitary adenoma. Not infrequently, as in our Case 1, the manifestations of Addison's disease are so indefinite as to escape notice or be misinterpreted. These patients often become drowsy, confused or comatose in the course of a few hours to days. The arterial hypotension and hypoglycemia which frequently accompany adrenal insufficiency may account for coma in some but not in all such cases. The syndrome of adrenal insufficiency can probably be distinguished from acute necrosis or hemorrhage in a pituitary adenoma by a more gradual onset of symptoms, by an absence of cranial nerve abnormalities, and by a normal CSF. The necessity of a careful assessment of the endocrine status prior to operation in those cases where pituitary apoplexy is quite certain requires no elaboration.

To differentiate between the syndrome caused by a massive extrasellar extension of a pituitary tumor with a secondary temporal lobe pressure cone, and that due to hemorrhage or necrosis in a pituitary adenoma may be difficult if not impossible. Coma, stiff neck and oculomotor palsy are common to both conditions. A temporal lobe pressure cone might be expected to occur only after embarrassment of the temporal lobe by the encroaching adenoma had announced itself by antecedent symptoms. Assuming, however, that evidence of a temporal lobe extension was at hand, it might be impossible to distinguish between these acute complications. Under such conditions lumbar puncture would be dangerous and perhaps indecisive. Prompt neurosurgical intervention would be justified in selected cases.

A convulsive state in cases in which there is an extension of the adenoma into a temporal lobe or the post-convulsive sequelae could conceivably be a source of difficulty in the differential diagnosis of the complications under discussion. A careful history of the illness, close observation of the patient and examination of the CSF should provide the necessary clues to the mechanism of symptom production.

E. *Clinical Management and Prognosis*

It is evident from cases here cited that in some instances the apoplectic insult will prove swiftly fatal. The outlook for patients surviving the initial impact of the illness will remain grave, and treatment at this critical juncture should be vigorously supportive. In addition to the application of conventional measures, special watch should be kept for the appearance of endocrinal deficiencies for which replacement therapy is possible. Even under an adequate supportive regime some patients will fail to rally but others will reach a plateau of partial improvement from which some of them may be rescued.

The possibility of continued and eventually complete, spontaneous recovery must be given first consideration. That it may occasionally be realized is indicated by probable examples reported, one each, by Cairns and by Jefferson. It should, therefore, be stressed that as long as the clinical course moves in the direction of recovery, treatment should remain conservative. X-ray therapy should not be advised after pituitary apoplexy because radiation would be too apt to provoke additional acute degenerative changes. If the patient makes a full clinical recovery it is doubtful whether the risk of an operation undertaken to prevent recurrence of apoplectic attacks should be assumed.

Unfortunately, our pathological material suggests that few patients will make spontaneous improvement beyond a condition of partial and temporary betterment. An extensive though largely circumscribed extravasation of blood at the infundibular region, or a post-infarction swelling of the extrasellar portion of the adenoma would doubtless give rise, in most cases, either to paralytic effects upon adjacent parts of the brain, or to blockage of CSF pathways and progressive internal hydrocephalus. Operation would then become necessary not in any prophylactic sense, but for relief of chronic headache, hemiparesis, impending stupor, papilloedema or threatened optic atrophy. The principal objective would be that of evacuating necrotic and hemorrhagic material so as to remove the pressure from neural elements and/or the obstruction to the free flow of CSF. Admittedly, surgical attack upon an adenoma that has escaped from the confines of the sella is a formidable undertaking, but if due respect is paid both to the hypothalamus, and to the remnants of the hypophysis, the results of intervention may well be gratifying. Indeed, of 3 operations thus far reported as having been done for relief of apoplectic syndromes (Dott, Bailey and Cushing,[5] Voss,[13] Sosman[11]), 2 were highly successful.

It is important to emphasize that if the acute symptoms here described overtake a patient who shows either the stigmata of acromegaly or those of pituitary insufficiency, the diagnosis of adenomatous apoplexy is tenable but only presumptive. In either instance the acute features of the illness might with equal plausibility be ascribed to the occurrence of a meningocerebral hemorrhage stemming, for example, from the rupture of a berry-aneurysm. Therefore, for the certainty of diagnosis which is requisite for operative treatment, intracranial vascular malformations should be excluded by angiography. This study might also provide valuable data regarding the suspected extrasellar extension of the pituitary lesion; it would not, however, obviate the need for pre-operative ventriculography. Use of the latter procedure has been urged by writers (Jefferson[6], White[14]) who have concerned themselves with the surgical treatment of the extrasellar adenomas that have not undergone acute degenerative changes. Similar indications for its use would obtain in planning operative exposures for the lesions under discussion.

SUMMARY

The clinical and pathological findings in 5 cases of adenoma of the pituitary body are presented. The unique feature of these cases is that hemorrhage into and/or extensive necrosis of the adenoma was responsible for the sudden onset of a fairly uniform syndrome consisting of drowsiness, stupor or coma, headache and stiff neck, ocular palsies and sometimes amblyopia or hemiparesis. Some abnormality of the CSF, usually blood or xanthochromia, though sometimes only slight pleocytosis or an elevated protein, was invariably present. These acute retrogressive changes are believed to be related to an excessively rapid growth of some pituitary adenomas. The necessity of correct diagnosis is emphasized. Several conditions that might prove troublesome in the differential diagnosis are discussed. These include crises of adrenal insufficiency, a temporal lobe pressure cone, and convulsive states. A program for the clinical management of cases of pituitary apoplexy is submitted.

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CASE REPORTS AND TECHNICAL NOTES

MENINGIOMA AND OLIGODENDROGLIOMA ADJACENT IN THE BRAIN

CASE REPORT

HARVEY GASS, M.D., AND WILLIAM P. VAN WAGENEN, M.D.
Department of Surgery, Neurosurgical Division, Strong Memorial Hospital, and The University of Rochester School of Medicine and Dentistry, Rochester, New York

(Received for publication November 10, 1949)

We wish to chronicle the present record of two tumors in the same patient, a meningioma and an oligodendroglioma, not only on account of their co-existence but because of the striking feature of their juxtaposition in the same hemisphere.

The literature on multiple primary brain tumors has been brought up to date in three recent reports.[1,3,4] Ours is the seventh recorded case of glioma and meningioma in co-existence. In none of the previously reported cases was there a combination of oligodendroglioma and meningioma. Although in Feiring and Davidoff's case[3] both tumors were recognized in life, the second one that appeared was a glioblastoma multiforme and the patient expired 2 months after the second craniotomy. In our case gross total removal of both tumors was accomplished at a single operation with a good prognosis for many years of normal life without serious deficit.

CASE REPORT

S.M.H. #293004. E.D., a 56-year-old right-handed woman, was admitted to Strong Memorial Hospital on Mar. 3, 1949, without known antecedent injury. In 1935 grand mal seizures developed, which occurred once or twice weekly until 1942. These began by her head jerking to the left and the left arm flinging outward. Following spinal anesthesia in 1942 for pelvic plastic repair, she was free of seizures until a similar one occurred in August, 1948 and again on 3 successive days 2 weeks prior to admission.

During the seizure of August, 1948, she suffered a laceration of the left side of her forehead and a fractured left shoulder. One month later when her arm was taken out of the cast she first became aware of weakness and swelling of the entire left upper extremity. She then also began to notice that her left leg was heavy so that she had difficulty when walking in keeping up with other people.

She denied paresthesias or visual symptoms at any time. Headaches, vertigo and vomiting were absent. She felt that of late she had been prone to forget things easily. There was no history of other change in the mental sphere.

Examination. A complete left hemiparesis was present, being least in the face, with slight spasticity of the left thigh muscles and hyperactive tendon reflexes in the left upper extremity. No abnormal reflexes were obtained in either lower extremity. Abdominal reflexes were not elicited. Stereognosis, touch, localization and two-point discrimination were intact, but several errors were made in recognition of number writing on the left hand. The optic fundi and visual fields by gross confrontation were normal. She was unable to subtract 7 from 100 serially correctly, and there were errors in simple calculations. Throughout the interview her attention was apt to wander.

Laboratory Studies. Blood studies, urinalysis, and stool examinations were all within normal limits.

Roentgenograms of the chest were normal. Roentgenograms of the skull (Fig. 1) revealed a fairly extensive area of calcification lying in the right frontoparietal lobes. It measured 4×5

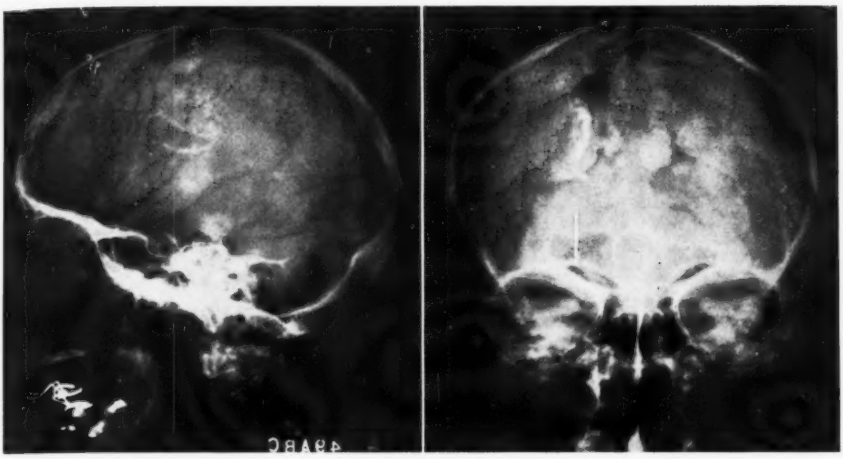


FIG. 1. Calcification of oligodendroglioma, lateral and anteroposterior views. Thickening of inner table of calvarium overlying calcification is evident in AP view.

cm. in the lateral view. This extended upwards quite close to the surface of the brain and there was a questionable bony change of the inner table superficially.

Operation. On Mar. 7, 1949, under local novocain anesthesia supplemented with intravenous sodium pentothal and with the patient in the sitting position, a right frontoparietotemporal flap was turned down. The brain was not under any particular increase in tension. The bone flap was attached to the dura in the region of the posterior frontal lobe and approximately $1\frac{1}{2}''$ from the midline and showed evidence of having been invaded by tumor.

The dura was reflected, the portion that was attached to bone and which was also attached to the underlying brain being encircled and left fixed to the subjacent tissue. This tis-

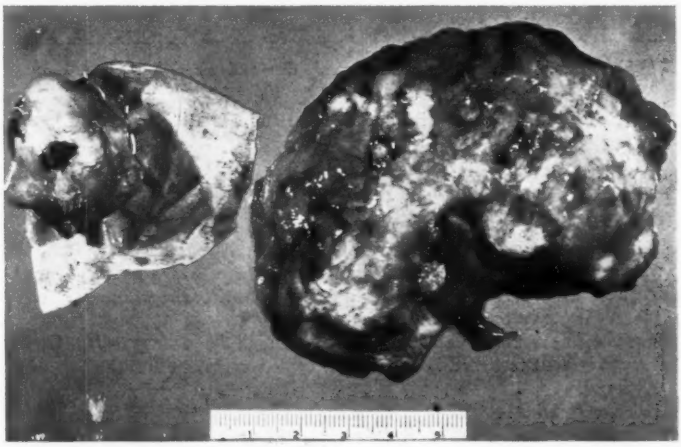
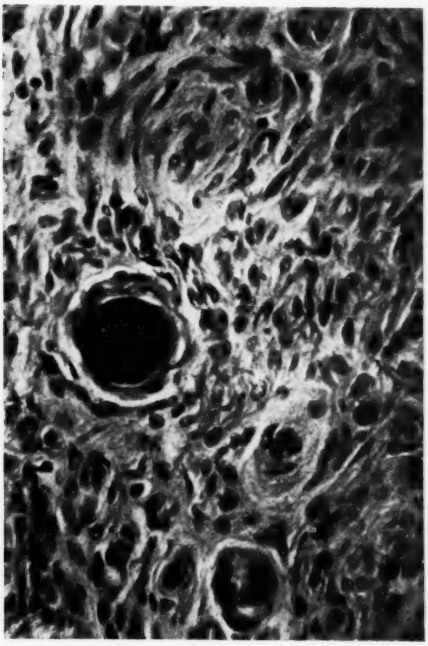


FIG. 2. Gross appearance of meningioma (left) and oligodendroglioma after removal.

sue appeared grossly to be a surface meningioma, measuring $1 \times 1\frac{1}{2}''$ in diameter and was readily removed.

Beneath it, by needle puncture, could be felt a calcified lesion with extension to the surface of the brain. This mass, the size of a tennis ball, was totally excised. At the end of the dissection the longitudinal sinus, the falx cerebri and the anterior cerebral artery were fully exposed in the wound and the ventricle had not been opened. After covering the exposed cortex with a sheet of Gelfoam film and excising involved bone, the wound was closed. During the middle of the procedure she had one convulsion.



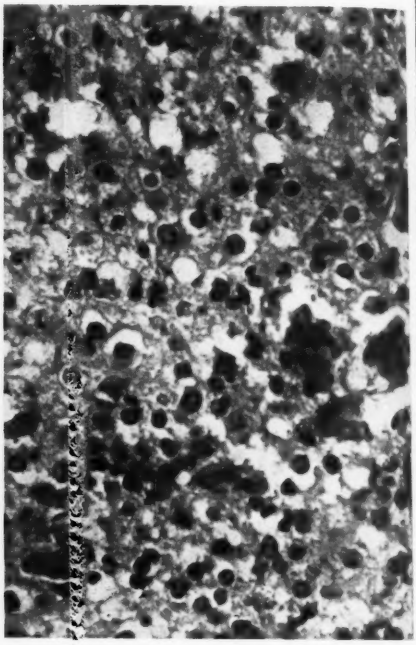


FIG. 3 (*left*). Meningioma showing psammoma bodies and whorl formation. Hematoxylin-eosin, ×360.

FIG. 4 (*right*). Oligodendroglioma. Perinuclear halos and calcifications are evident. Hematoxylin-eosin, ×360.

Postoperative Course. On the 1st day her vital signs were well maintained. She was alert, rational and eating. There was feeble movement of the left lower extremity, none in the left upper, and there was a slight left facial weakness. On the morning of the 2nd day, she became semi-comatose, responding only to deep pain; the right pupil was larger than the left. The scalp flap was reopened at the anterior portion of the wound which allowed a gush of bloody CSF, air, and a small clot to escape. After thorough irrigation the wound was closed, leaving a drain in place. Shortly after this the patient awoke.

Her course was uneventful although at first a left hemiplegia was present. On the 5th postoperative day movement returned in her left lower extremity, and on the 9th she was able to move her left hand.

She was discharged on the 12th postoperative day. At that time she was able to walk a few steps alone. The strength and use of the left upper extremity was increasing day by day and

there was no sensory deficit. Fields were normal by gross confrontation and the discs were flat. The decompression was soft and the wound well healed. Dilantin, 0.1 gm. 3/day, was prescribed. She had had no postoperative seizures.

Pathological Report. The gross diagnosis of meningioma for the superficial tumor (Figs. 2 and 3) was confirmed, but the diagnosis on the deeper calcified mass was oligodendroglioma (Fig. 4).

DISCUSSION

The adjacency of the two tumors in this case invites speculation regarding the incitement of growth of one by the other. For two reasons, it would appear that the meningioma was the younger of the tumors. First, it was smaller and second, the presence of the calcification in the oligodendroglioma would suggest its existence for a considerable length of time, presumably as far back as 1935 when seizures first began. The size of the meningioma was hardly compatible with a duration of 14 years. The oligodendroglioma reached a point near the surface of the brain and it would appear likely that it could have acted as a source of irritation to arachnoidal or dural cells overlying it, leading to local proliferation of these elements and final independent tumor formation at the site of the proliferation. The high incidence of head trauma in Cushing's[2] series of meningiomas suggests a similar irritative etiological role in many of these tumors. The appearance of the second tumor in our patient may possibily also have depended to a certain extent upon a greater inherent susceptibility to neoplastic growth.

It is of some interest in her clinical course that following a pelvic operation which was done under spinal anesthesia she was entirely free of her previous frequent seizures for 6 years.

SUMMARY

A case report is presented of two tumors, a meningioma and an oligodendroglioma, lying in the same hemisphere in juxtaposition in a 56-year-old woman who had had epilepsy for 14 years. Gross total removal of both was accomplished at one operation and the prognosis for prolonged recovery appears good. Because of the probability that the oligodendroglioma was the older of the two tumors, the suggestion is made that it served etiologically as an irritant leading to the development of the meningioma over it.

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CHRONIC EXTRADURAL HEMATOMA

IRA J. JACKSON, M.D.,* AND THOMAS J. SPEAKMAN, M.D.

Department of Neurology and Neurosurgery, McGill University, and the Montreal Neurological Institute, Montreal, Canada

(Received for publication December 7, 1949)

The classical syndrome of acute extradural hemorrhage has been repeatedly emphasized in the literature.[2,3,4,8,9,10,13,14] The reported lucid intervals have varied from a few moments to 31 days. However, all patients have ultimately presented focal neurological signs and evidence of increased intracranial pressure. Thus extradural hematomata are considered to occur or terminate as an acute process. It is for this reason that we wish to report a patient with a large extradural hematoma existing in a chronic state.

CASE REPORT

A large chronic encapsulated extradural hematoma was encountered in the following patient† during a craniotomy performed 36 days after his head injury for the presumptive diagnosis of intracerebral hematoma.

L.L., a healthy 23-year-old male laborer, was struck in the right temporoparietal region by a piece of pulpwood on March 8, 1949. He was rendered unconscious for a period of 5 to 10 minutes, and dazed for the following hour. During the remainder of the day he experienced severe frontoparietal headache with nausea and vomiting. The next day he was admitted to his local hospital, complaining of headache, nausea and vomiting. Normal neurological findings and a clear colorless CSF were noted. However, nausea and vomiting continued for 48 hours when relief was obtained by giving 500 cc. of 5 per cent glucose in saline intravenously. The patient was asymptomatic for the following 4 days, but on March 14, generalized headache, nausea, and vomiting recurred. Lumbar CSF pressure was normal but symptoms were alleviated by the puncture. He was asymptomatic for another 2 weeks, but from March 28 until April 11 lumbar punctures were performed several times for relief of headache and vomiting. Lumbar CSF pressure was thought to be elevated on only one occasion but no manometric readings were available.

On admission to the Montreal Neurological Institute, April 11, 1949, 34 days after his injury, the patient was asymptomatic and presented no abnormal neurological findings. The only relevant finding was a mild subjective tenderness of the scalp over the right temporoparietal region. The lumbar CSF was clear and colorless under 120 mm. pressure, containing no cells and 42 mg. per cent of protein. No fracture was demonstrated by roentgenograms of the skull. An oxygen encephalogram performed on April 12, 1949 showed the anterior wall of the right lateral ventricle flattened and displaced backwards 1.5 cm. (Fig. 1A). The normal convexity of the anterior end of the ventricle was replaced by a slight concavity, and the septum pellucidum anteriorly measured 6 mm. to the left of the midline. The left lateral ventricle did not fill with oxygen.

With a tentative diagnosis of an intracerebral hematoma, a twist drill biopsy of the right frontal lobe was performed. No gross pathological lesion was encountered, but microscopic sections of a brain worm showed hypertrophied glial cells, new vessels and some endothelial overgrowth. Since the left lateral ventricle had not been visualized, a ventriculogram was performed through bilateral frontal twist drill holes. Both lateral ventricles and the third ven-

* Now at the University of Texas Medical School, Galveston, Texas.

† This patient was referred to Dr. William V. Cone by Dr. J. King of Cornerbrook, Nova Scotia.

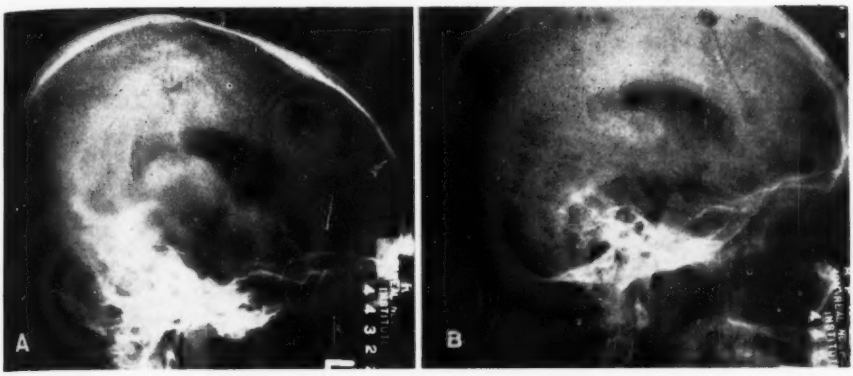

FIG. 1. (A) Preoperative ventriculogram. This brow-up left lateral view shows the posterior displacement and flattening of the anterior horn of the right lateral ventricle. (B) Encephalogram made 17 days after operation. This corresponding brow-up left lateral view shows the return of portion one of the right lateral ventricle to its normal contour except for a minimal elevation of the floor.

tricle were filled with oxygen, confirming the impression gained by the encephalogram, namely a right frontal space-occupying lesion (Fig. 1A).

Operation. A right frontal osteoplastic craniotomy was carried out under general anesthesia on April 13, 1949, 36 days after the injury. A large encapsulated extradural hematoma was found in the anterior fossa (Fig. 2). It extended to the midline superiorly and inferiorly, and to the lesser wing of the sphenoid posteriorly. The capsule, consisting of fibroblasts and collagen, measured about 5 mm. in thickness. The lateral wall was firmly adherent to the inner table of the skull and the medial wall was coextensive with the dura. The hematoma consisted of 50 to 75 cc. of large blood clots and black-brown fluid. The frontal lobe and overlying dura were displaced posteriorly 3 to 4 cm. from the anterior cranial vault. After the hematoma had been evacuated and the capsule dissected away, a small fracture was found in the middle of the lesser wing of the sphenoid. No source of active bleeding was discovered. At the conclusion of this procedure the brain was expanded to its normal contour by means of the introduction of Elliott's artificial CSF through the lumbar subarachnoid space.[7]

FIG. 2. Photograph of chronic right frontal encapsulated extradural hematoma taken at operation. Part of the outer wall of the capsule had been removed with the free bone flap. Note the edge of the medial wall of the capsule attached to the dura and the amount of posterior displacement of the dura overlying the right frontal lobe.

The *postoperative course* was uneventful

except for some headache and vomiting during the first 36 hours. The CSF dynamics were normal. An oxygen encephalogram done 17 days postoperatively showed only minimal elevation of the floor of the anterior portion of the lateral ventricle (Fig. 1B). An EEG on May 13 revealed minimal slow wave activity over both frontal poles.

DISCUSSION

This patient with a chronic extradural hematoma is unusual because of the long interval of 36 days between trauma and operation, and the absence of abnormal neurological findings or impairment of vital functions. The longest previously reported interval before operation was 31 days.[2] In that patient progressive focal neurological findings and impaired sensorium necessitated surgical intervention. Although our patient had occasional lumbar punctures with immediate relief of headache and vomiting, at no time did his clinical picture make operation imperative. Indeed, in the absence of localizing signs, operation was performed, on the basis of encephalographic findings, to exclude tumor and to evacuate the suspected intracerebral hematoma.

The area affected could perhaps account for the absence of neurological findings in the presence of such a large space-occupying lesion. However, in 3 cases of *acute* anterior fossa extradural hemorrhage,[1] localizing cranial nerve and pyramidal tract signs were present. The absence of signs must therefore be explained by the source and rate of the bleeding. The most common source of hemorrhage in extradural hematomata is one of the branches of the meningeal artery.[11] Other sources less frequently seen are meningeal veins,[5] dural sinuses, and diploic and emissary vessels. Those from the main anterior branch of the middle meningeal artery or from the superior petrosal sinus usually run a more or less fulminating clinical course. It would seem probable that the bleeding occurred from a small vein or artery and took place slowly over a period of at least a few days. The slowly expanding clot could be expected to act much like a neoplasm of this region. This would explain the lack of impairment of vital functions, the normal neurological findings, and the normal CSF dynamics.

The presence of a capsule surrounding degenerating and organizing blood products is not unusual in subdural and other hematomata, but, heretofore has not been described for extradural hemorrhage. This is presumably because of the usually short time interval between hemorrhage and operation or death. However, Gurdjian, in a patient operated upon 31 days after the original injury, made no mention of capsule formation.[2]

It is interesting that in clinical series of head injuries, extradural hemorrhage is encountered in 2 to 5 per cent of cases.[9] Postmortem series, on the other hand, report extradural hematomata sufficiently large to cause death in 12 to 20 per cent of craniocerebral injuries.[6,12] While this may have several explanations, it indicates that many extradural hemorrhages still go unrecognized. Doubtless, some patients live without operation and the collections subsequently organize or absorb. Others, as this patient demonstrates, may become encapsulated and persist, producing the brain changes described above. The later stage of this process would be extensive gliosis and cerebral atrophy which, depending upon the location, could result in a profound neurological and psychological deficit.

SUMMARY

A patient with a chronic encapsulated extradural hematoma operated upon 36

days after injury is reported. The reason for the absence of neurological signs in the presence of a large extradural lesion may be explained by the location and source of bleeding. Investigation for this lesion should be made in post-traumatic cases with symptoms suggesting increased intracranial pressure, even in the absence of focal neurological signs. Early surgical intervention is indicated to prevent pathological changes in the subjacent brain.

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OSTEITIS FIBROSA CYSTICA LOCALISATA OF THE SKULL

John Paget, M.D.,* Geoffrey Fricker, M.D., and Adrien Ver Brugghen, M.B.

Departments of Neurosurgery and Radiology, Presbyterian Hospital, Chicago, Illinois

(Received for publication December 21, 1949)

The localized form of osteitis fibrosa cystica in the skull is a rare disease. It is with this entity that the present case report will be concerned. In the localized form, it must be borne in mind that there is no pathological change in the parathyroid glands nor are there associated changes in the blood calcium, phosphorus or phosphatase. The condition is mentioned only briefly in most treatises, the most complete being that of Chorobski and Davis.[2] In a rather general discussion of bone cysts in the skull, they describe one case of the localized form of osteitis fibrosa cystica. Geschickter and Copeland[4] consider some solitary bone cysts a form of osteitis

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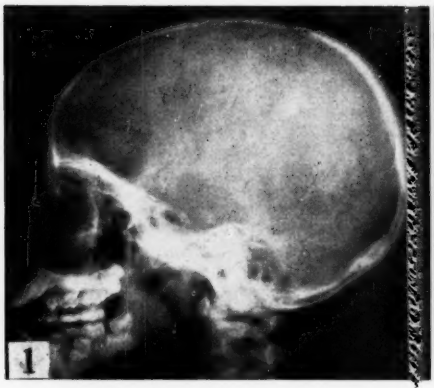
fibrosa cystica. These are found almost without exception in the long bones rather than in the skull, and the age incidence is 6 to 42 years. Compere[3] writes in some detail of osteitis fibrosa cystica localisata and stresses the fact that the body chemistry remains unchanged. Albright[1] in his recent monograph describes the localized form of the disease but makes no mention of its occurrence in the skull. The following case report concerns a 13-year-old girl who had been seen in the Clinic some years before and whose plain skull x-rays were normal at that time.

REPORT OF CASE

A 13-year-old colored female was first admitted to the Presbyterian Hospital in August, 1944. At that time, she had epileptic seizures which began in 1940. These were mild in nature and of the petit mal variety. The attack lasted 30 seconds. There was no aura nor loss of con-



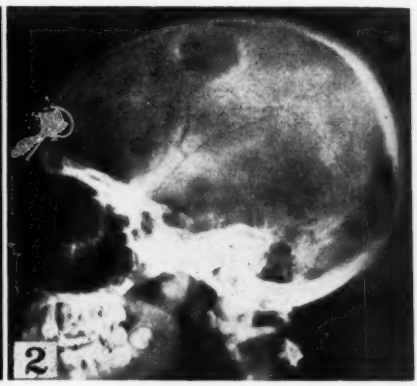


FIG. 1. This shows film of skull made prior to the development of the cystic lesion.

FIG. 2. Film taken 5 years later, showing the cystic area of bone destruction in the frontoparietal skull area.

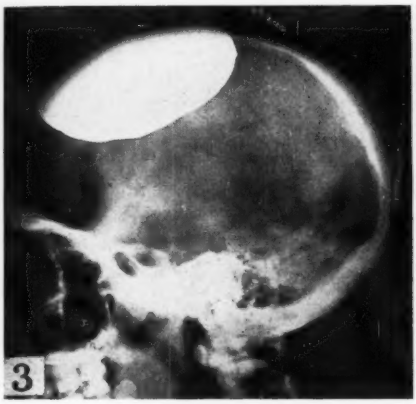
sciousness and the convulsive movements, clonic in type, involved all extremities. The birth and developmental histories were normal.

Examination. Temperature 99.2, pulse 85, respirations 20, B.P. 110/70. She was a normally developed 8-year-old colored female. The throat was injected. There was no evidence of abnormality of the skull. She had mild bilateral nystagmus and her gait was ataxic. The remainder of the neurologic findings were within normal limits. The ataxia and nystagmus had cleared by the time the patient was ready to be discharged from the hospital. Roentgenograms of the skull at this time were normal. The lateral film is illustrated in Fig. 1. A pneumoencephalogram showed normal ventricles and normal cortical markings. No EEG was done during this admission. She was placed on phenobarbital gr. ss b.i.d. and discharged.

Interval History. During the next 5 years, the child got along fairly well. She had occasional seizures similar to those described at the time of her first admission. On May 25, 1949, gastroenteritis developed, presumably due to food poisoning, and during this illness her seizures became more frequent. Once again too, she became somewhat stuporous, nystagmus developed and her gait was ataxic. All of these cleared after several days. During this time she was seen in the Clinic and new x-rays of the skull were taken. These showed a single, rounded area of bone destruction with a sclerotic margin in the left frontoparietal area (Fig. 2). Because of this finding, it was felt that the patient should be readmitted to the hospital for further evaluation.

2nd Admission. Temperature 98.6, pulse 72, respirations 20, B.P. 120/80. The girl was somewhat retarded mentally. The only positive neurologic finding was an increased right patellar reflex. Rbc. was 3,928,000, hb. 11.3 gm., wbc. 6,800. Urinalysis was negative. Blood calcium was 10.3 mg./100 cc., blood phosphorus 4.9 mg./100 cc. and alkaline blood phosphatase 10.9 Bodansky units. The EEG was interpreted as being within normal limits for her age. A repeat pneumoencephalogram showed normal ventricles and cortical markings. Intravenous pyelograms and x-rays of the chest, spine and long bones were all normal.

Operation. Because of the questionable nature of the lesion and particularly since the question of metastatic tumor had been raised, it was decided that surgical exploration with removal of the area of skull should be carried out. On Aug. 2, 1949, an ordinary plastic scalp flap was turned over the area of cystic bone. The lesion was seen to be elevated above the surrounding cortex and measured 4.5 cm. in diameter. Two burr holes were placed opposite one another at the periphery of this lesion and these were then connected in circular fashion with the DeVilbiss rongeur. The inner table showed no evidence of erosion nor was there any



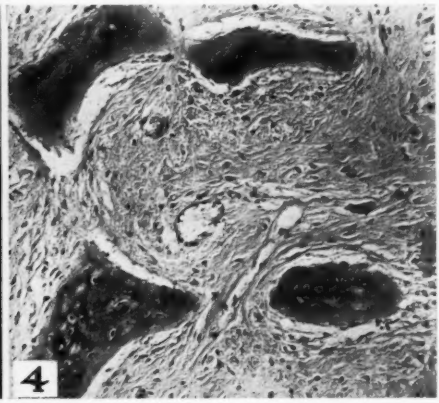


FIG. 3. Postoperative film showing tantalum plate in place.

FIG. 4. Photomicrograph showing spicules of bone surrounded by bulky connective tissue in various stages of development.

indication that the cyst was exerting pressure on the underlying cortex. A tantalum plate was inserted (Fig. 3) and the wound then closed in layers. Sutures were removed on the 9th postoperative day and the wound was well healed.

Pathological Report. The specimen consisted of a roughly circular segment of hyperostotic bone measuring 4 cm. in diameter and 1 cm. in thickness. It contained portions of two burr holes at the periphery and a suture line traversed the center. On microscopic examination (Fig. 4) there was evidence of atrophy of bony spicules and the formation of some new bone. The marrow was replaced by fibrous connective tissue, some of which was vascular. This tissue was in various stages of development. Microscopic diagnosis: Osteitis fibrosa cystica.

COMMENT

Localized osteitis fibrosa cystica in the skull is a rare disease. The x-ray picture is that of an area of bone destruction with a surrounding sclerotic margin. It must be differentiated from metastatic tumor and it was this question in particular in the present case that demanded surgical investigation. Of special interest was the availability of x-ray studies of the skull made prior to the development of the lesion.

It was also pointed out that the changes in calcium, phosphorus and phosphatase that occur in the generalized form of osteitis fibrosa cystica are not seen in the isolated type. Finally, a brief description of the operative procedure carried out was presented and the microscopic picture of the disease described.

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SYSTEMIC BLASTOMYCOSIS WITH SPINAL CORD INVOLVEMENT

CASE REPORT

Robert C. Greenwood, M.D., and Harold C. Voris, M.D.
Mercy Hospital and Loyola University Clinics, Chicago, Illinois

(Received for publication December 27, 1949)

This case is reported because the unusual clinical course and pathological aspects make it important not only in the differential diagnosis and treatment of surgical conditions of the spinal cord but also as a rare manifestation of an already uncommon grave disease. Blastomycosis usually takes either a cutaneous or a systemic course. The cutaneous type tends to heal, whereas the systemic type is characterized by chronicity, and widespread infection in the lungs, subcutaneous tissues, bone, and internal organs. Prognosis in this type is extremely grave.

Blastomycosis itself is relatively rare, since up to 1939 Martin and Smith[4] reviewed 347 proved and presumptive cases. In only 16 of these cases was the central nervous system involved. The usual course of such involvement is that single or multiple abscesses develop in the cerebrum, cerebellum, and brain stem, with a basilar purulent meningitis as a part of the generalized systemic variety. However, in 1940, Craig, Dockerty, and Harrington[1] reported an intravertebral and intrathoracic extradural mass that simulated a dumb-bell tumor. It proved to be a blastomycotic lesion and it is probably the first case report of such a lesion simulating a tumor in the spinal canal. In Friedman and Signorelli's[2] case the blastomycosis involved only the meninges of the brain and spinal cord. Detailed descriptions of the usual course and pathology of both types of blastomycosis have been described by Montgomery and Ormsby,[5] Wade and Bel,[8] and Stober.[6] With regard to the identification and isolation of Blastomyces dermatitidis and differential features of mycotic lesions, Stoddard and Cutler,[7] Martin and Smith,[4] and Hassin[3] have covered the important points.

REPORT OF CASE

J.M., a 40-year-old white woman, referred by Dr. L. Kratz, was admitted to Mercy Hospital on March 7, 1946, with a diagnosis of myeloma or spinal cord tumor. Her chief complaints were numbness beginning in the calves of the legs and reaching the waist, and progressive

weakness of both legs with difficulty in walking. Both complaints had developed over a 3-week period prior to admission. For 3 years, she had experienced mild mid-dorsal pain in the back that was relieved by ordinary analgesics and was not aggravated by coughing or straining. She had had a fractured right rib 8 years before, and blastomycosis of the skin of the face and upper extremities 4 to 5 years prior to admission. Biopsy of the skin at that time was reported as blastomycosis. The skin lesions healed rapidly and she was apparently well until the onset of the present illness.

Examination. There were several small white scars on the left cheek of the face. Neurological findings were: hypesthesia of the lower limbs and trunk with a level at the 7th thoracic dermatome, absent vibration sense in the left leg, weakness of the trunk muscles and lower limbs, equivocal Babinski signs, and ankle clonus bilaterally.

Roentgenograms of the spine showed erosion of the pedicles of the 6th and 7th dorsal

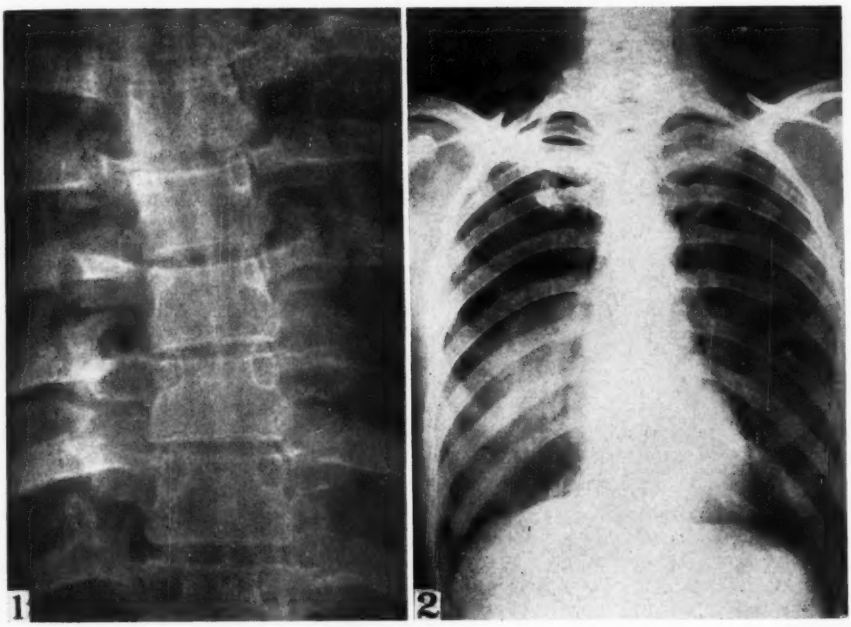

FIG. 1. The X-ray of the dorsal spine revealed erosion of the pedicles of the 6th and 7th dorsal vertebrae, with a punched-out area of decreased density in the head of the right 7th rib.

FIG. 2. The patient had no complaints referable to the chest. The film was reported to have a light homogeneous density in the right lung extending out from the root.

vertebrae with impairment of those of the 8th body. A discrete punched-out area, about 1 cm. in diameter, was found in the head of the 7th rib on the right side (Fig. 1). A routine chest plate was reported to have a light homogeneous density about 6 cm. in diameter, irregularly outlined, but extending out from the right lung root and fading in the parenchyma of the right lung (Fig. 2). Urine analysis was normal. Rbc. 3,850,000; Hb. 74 per cent; Wbc. 7,200 with 54 per cent polys, 40 per cent lymphocytes, 1 per cent eosinophils, and 3 per cent monocytes.

Operation. On March 9 an extensive laminectomy and decompression of the spinal cord with partial removal of an extradural granuloma was carried out. Purulent material exuded from a cavity in the articular process of the 7th rib. The upper and lower limits of the gran-

uloma were uncovered opposite the 3rd and 8th vertebral bodies respectively. The lesion was thickest on the right side and opposite the 6th and 7th bodies, where it was 1 cm. thick on the dorsal surface.

The *postoperative course* was uneventful, and she was dismissed on April 1, 1946, with moderate improvement in neurological function in that the hypesthesia had receded and she could walk with assistance.

Pathological Report. The surgical specimen was a granuloma with single and budding forms of a pathological yeast rather sparsely distributed. Culture of the fresh material on Sabouraud's media yielded a growth of Blastomyces dermatitidis.

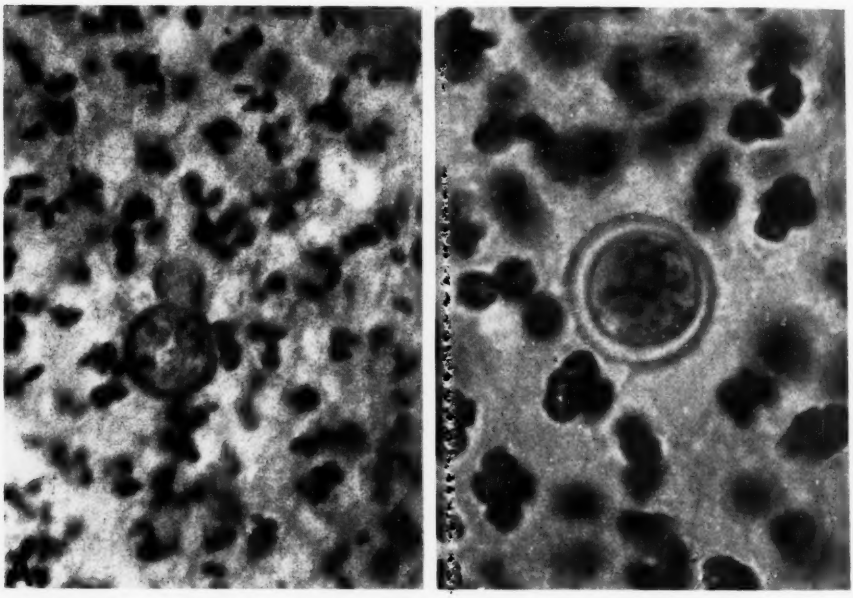

FIG. 3. Microscopic abscess. (A) Single budding double-contoured organism surrounded by polymorphonuclear leucocytes (×430). (B) Double-contoured organism (×930). The organism was found to be Blastomyces dermatitidis when cultured. (Hematoxylin and eosin stain.)

Further Course. Soon after dismissal from the hospital, the neurological complaints recurred, with loss of visceral sphincter control. She was readmitted on May 10, 1946. She had an analgesic level at the umbilicus, spastic paraplegia with mass reflexes of both legs, and an atonic, incontinent bladder.

Lumbar puncture revealed a complete block. Only 5 cc. of CSF could be obtained; this contained 8 cells/c.mm., 300 mg. per cent total protein, and numerous round to oval bodies with a doubly refractile ring found on direct smear.

The patient received nine deep X-ray treatments while in the hospital, but she became very depressed and desired to return home. She was dismissed on June 10, 1946.

Subsequently a small draining sinus developed in the dorsal region of the back. During June 1946, she was examined at another institution where cultures from the sinus were reported to be those of Blastomyces. The course of her illness was progressively downward, and still later she was hospitalized in her home city, where Blastomyces were again cultured from the sinus. She died about October 1946.

Autopsy. There were chronic inflammatory granulomatous lesions in the lungs, and thickened dura along the spinal cord, with elevated patches of grayish tissue on the dura for about 14 cm. of the thoracic region. About 20 cm. from the conus, this thick gray tissue entirely encircled the cord. The dura was about 1 mm. thick in this region.

Microscopic Examination. The tissue at the mid-dorsal region in the extradural space contained numerous foci of polymorphonuclear neutrophils surrounded by a zone of macrophages and fibroblasts. Multinucleated cells were numerous in some areas as well as new capillaries. Rather sparsely distributed were round to oval doubly refractile bodies, some single and others with a single bud in these foci of neutrophils. The dura was adherent to this tissue and very thick, with numerous capillaries surrounded by small round cells and polymorphonuclear neutrophils.

The spinal cord itself showed no changes other than softening and degeneration of its columns following vascular changes from the cuff of granuloma about it. There were no significant findings in the brain.

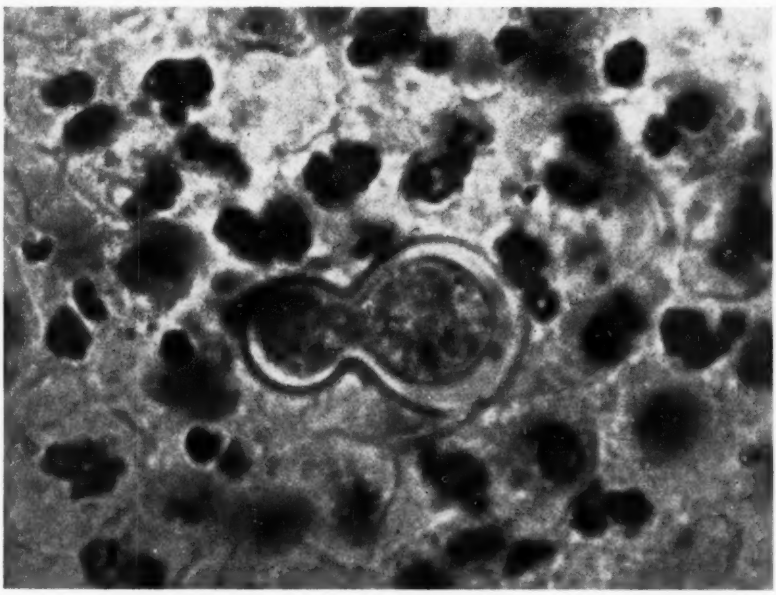

FIG. 4. Double-contoured organism in process of budding. The toluidine blue stain failed to reveal the spines and capsule that are characteristic of Torula. (Toluidine blue stain, ×1160.)

Diagnosis. It was believed that the microscopic abscesses, the predominance of polymorphonuclear neutrophils and chronic inflammation were features that differentiated the lesions from tuberculosis. A pathological yeast was considered more likely to be the causative agent since different laboratories isolated the organism and reported it to be Blastomyces dermatitidis. This organism was further differentiated from Torula histolytica by staining the sections with toluidine blue, which brings out the typical capsule and its numerous spikes, spines, or spicules of the Torulae.[3] This morphology was not demonstrated in the present case; the organisms remained oval or round with a doubly refractile ring (Figs. 3 and 4). The presence

of single budding forms differentiated this yeast from Coccidioides immitis; the latter organism never exhibits budding. A peripachymeningitis with extradural abscesses simulating spinal cord tumor, due to Blastomyces dermatitidis, was probably the primary lesion.

COMMENT

This patient was apparently cured of her skin lesions of blastomycosis. After a history of mild non-progressive pain in the mid-dorsal region for about 3 years, there rapidly developed a neurological deficit that caused her to seek attention. In the absence of signs and symptoms of systemic involvement, a diagnosis of a spinal cord tumor was very probable. The clinical manifestations and roentgenograms seemed to confirm this diagnosis. Also, the lack of systemic involvement by the Blastomyces made it difficult to interpret the discrete punched-out area in the head of the 7th rib on the right side. Dr. H. E. Potter in 1914 emphasized the sharp line of demarcation between bone apparently intact and areas of total necrosis.[6] He felt that such a marked localizing destruction was so constantly present in ordinary blastomycotic lesions, that a search should be made for Blastomyces. However, the lesions are most distinct and numerous in the long bones. The localized nature of the lesions seemed, therefore, to justify surgical exploration.

SUMMARY

An unusual case of systemic blastomycosis is reported. A difficult problem in differential diagnosis was presented by the clinical manifestations of a localized lesion in the spinal canal on the one hand, and the healed cutaneous blastomycosis without apparent evidence of systemic involvement on the other hand. The organism isolated by different laboratories was reported to be Blastomyces dermatitidis and its morphology remained constant when stained with toluidine blue. The previously described roentgenographic characteristics of the bone lesions in blastomycosis are emphasized.

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MENINGIOMA OF RECORD SIZE WITH UNUSUAL FEATURES

JAMES C. WHITE, M.D., ROBINSON LeR. BIDWELL, M.D.,
AND CHARLES S. KUBIK, M.D.

Neurosurgical Service and Department of Neuropathology, Massachusetts General Hospital, Boston, Massachusetts

(Received for publication January 20, 1950)

Intracranial tumours of record size occasionally merit a brief report, especially if they give rise to unusual complications. The following case history is of interest because the tumour was first visualized in the lateral ventricle in a child of $5\frac{1}{2}$ years and had reached the enormous size of 1353 grams when finally removed 10 years later. Inconclusive biopsy at the first operation led to failure in establishing the correct diagnosis, and prolonged refusal on the part of the parents prevented removal long after its slow growth and deposition of calcium salts had made the benign character of the tumour evident. Although complete removal of this meningioma was carried out successfully in two stages, epileptic seizures with mental deterioration followed and the patient finally died 6 months afterwards of hydrocephalus secondary to an unusual hygroma which developed in the huge cavity left after removal of the tumour.

A review of previous accounts of meningiomata of record size shows that the largest described by Cushing and Eisenhardt[1] weighed 310 grams. In their book they also cited an unpublished 341-gram meningioma removed by Hoen. Pilcher,[5] who summarized the literature in 1943, reported one of 374 grams, which he had removed, and mentioned a still larger one recorded by Davidoff.[2] In this case the weight of 835 grams included not only the tumour proper but an undetermined amount of cranial exostosis.*

CASE HISTORY

A $5\frac{1}{2}$-year-old boy first entered the Massachusetts General Hospital in 1936, with a history of intermittent vomiting and headaches which had begun several months after a fall downstairs at the age of 18 months. The baby had been unconscious for a few minutes, but had recovered without other immediate sequelae.

Examination revealed right internal strabismus and nystagmus. Awkwardness and dysynergia of the right arm with unsteadiness of gait suggested a cerebellar neoplasm. Motor power, sensation, and reflexes were normal. Ophthalmological findings were papilloedema of 5 D., reduction of visual acuity to 20/200, and contracted visual fields with a suggestive right homonymous hemianopsia. Skull films at this time showed evidence of prolonged increase in intracranial pressure.

At the time of ventriculography combined ventricular-lumbar pressures were recorded:

	R. ventricle	*L. ventricle*	*Lumbar space*
Initial pressure:	400	—	455
Colour:	Clear & colourless	Deeply xanthochromic	Slightly xanthochromic
Protein:	24	2280	708
Goldsol:	1122210000	3445555555	—

* Since this manuscript has been accepted for publication Dr. Eisenhardt has called our attention to a case report by C. W. Laymon in *Minnesota Medicine* for November, 1949 (*32:* 1132–1133). This records a giant meningioma which had invaded the right frontopartietal portion of the skull and expanded beneath the scalp to a "size of two grapefruits" and at post mortem weighed 2300 grams. The

Ventriculography showed the right lateral ventricle to be enlarged. Both it and the 3rd ventricle were displaced to the right. Only the posterior horn of the left lateral ventricle filled with air. This appeared to be enlarged with "a sharp concave anterior margin consistent with amputation of the ventricle in this area."

1st Operation. On Nov. 23, 1936 a left occipitoparietal craniotomy and exploration, carried out by Dr. John S. Hodgson, disclosed an encapsulated tumour which blocked the midportion of the lateral ventricle. Owing to the high degree of intracranial pressure and bleeding, it was felt that a radical attack on the tumour was out of the question, so only a biopsy was taken. It was not possible to close the bone flap, which had to be sacrificed to provide for decompression.

Microscopic Report. The specimen proved inadequate for a positive diagnosis. "The findings are probably consistent with polar spongioblastoma, although fibrils are more numer-

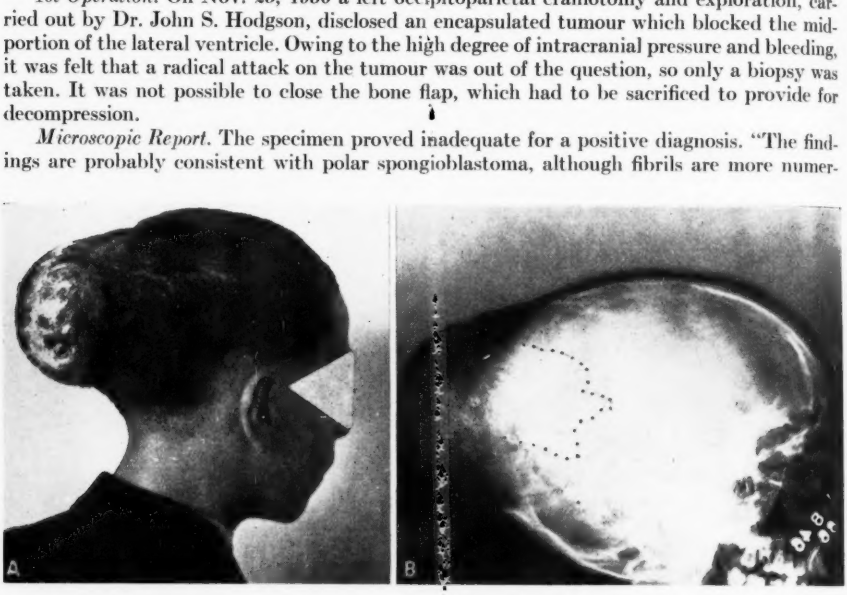

FIG. 1. (A) Appearance of patient and extracranial herniation of tumour just before its removal. (B) Left lateral plate of skull to show size of tumour from area of calcification. Dotted line marks intracranial extent of calcium deposits as seen in x-ray film.

ous and heavier than is usual. The wall of a cyst in an astrocytoma is another possibility."

Course. Postoperative convalescence was uneventful, but a cerebral hernia and a right homonymous hemianopsia rapidly developed. During the ensuing 10 years the hernia slowly increased. In 1937, except for the homonymous field defect, a mild degree of optic atrophy, and a 5-inch herniation, he was a normal appearing boy. Headaches and papilloedema had subsided. In 1942, however, there developed a right hemiparesis and convulsive seizures. These began with an epigastric aura and clonic movements of the right arm, which rapidly became generalized. Although right-handed and temporarily unable to talk after his seizures, there was never persistent aphasia. At our suggestion he was taken by his parents to Montreal, where he was examined by Dr. Wilder Penfield. Despite his advice and repeated efforts on our part, operation was refused by the parents. In 1946 the boy had to be taken out of school because of his seizures and was finally readmitted for operation at his own insistence in September, 1946.

Examination on 2nd Admission. The occipitoparietal herniation protruded a good 6 inches (Fig. 1A). There was a moderate degree of right-sided hemiparesis with increase in reflexes

specimen included the invaded portion of the skull and no mention is made of its intracranial volume. The patient was a 72-year-old recluse with a 7-year history of frontal swelling, 5 years of deterioration of mental faculties, and 2 years of weakness in the left arm with epileptic seizures.

and a reduction in sense of touch and astereognosis. Eye findings were right homonymous hemianopsia with macula splitting, optic atrophy, and no papilloedema. Visual acuity was reduced to fingers at 2 feet. Lumbar puncture gave an initial pressure of 225 mm.; protein was 124 mg. per cent. X-rays now revealed strands of calcification within the herniated tumour mass, which extended forward and medially nearly to the centre of the skull (Fig. 1B).

2nd Operation. Extirpation of the tumour was undertaken in two stages. On Jan. 14, 1947 the herniating portion of the fibrous calcified meningioma was excised. Except for the vascular capsule, which contained many veins the size of a lead pencil, no unexpected difficulties were encountered. Loss of blood, although profuse, was well compensated for by multiple transfusions. This portion of the tumour weighed 520 gm.

Course. The boy was discharged 3 weeks later and readmitted after 3 months in much the same condition.

3rd Operation. The remainder of the tumour, weighing over 833 gm., was resected on April 3, 1947. By turning down an additional cranial flap anterior to the pre-existent bony defect a good line of cleavage between normal brain and tumour could be developed. Beyond some difficult moments in securing the large vessels at the base of the tumour, the entire mass was resected without difficulty. This left a huge defect in the posterior half of the skull with the falx exposed at the midline and the tentorium at the base. The cavity was filled with Hartman's solution.

Course. The patient recovered without increase in his hemiparesis or aphasia, but he ran a febrile course (101–103°) for 18 days with a very bloody CSF. At discharge on April 25, 1947, he was continuing to have frequent minor seizures, during which he did not lose consciousness but was unable to speak.

4th Admission. His final hospital admission was on Sept. 10, 1947 in status epilepticus, from which he soon recovered on heavy anticonvulsant medication. At this time his scalp bulged with a large accumulation of fluid. On tapping the cavity the initial pressure was 140 mm. The fluid was xanthochromic and contained 10 lymphocytes with many crenated rbc.; protein 270; sugar 89 mg. per cent. Neutral phenolsulphonephthalein injected into the cavity was recovered in small amount in the lumbar subarachnoid space. During the following days 400 to 1,700 cc. of fluid were aspirated frequently from the cavity, only to reaccumulate within a few days. On Oct. 4, 1947 a lumbar puncture was done after 2 days of fever spiking to 104°. The protein content had now risen to 600 mg. and the cell count to 750 with 60 per cent polymorphonuclears, 25 per cent lymphocytes, and 15 per cent crenated rbc. The sugar had fallen to 33 mg. per cent. Repeated cultures were sterile. On treatment with penicillin and sulphadiazine the fluid cleared, but the patient continued to run a low-grade febrile course and gradually sank into coma. Death occurred on Oct. 26, 1947.

Pathological Examination. The surgical specimens removed at the last two operations formed a hard, lobulated, roughly ovoid tumour measuring 14×12.5×11 cm. and weighing 1353.5 gm. The outer surface was covered by a glistening, thin, fibrous capsule with numerous large veins. The cut surface was white, avascular, and exceedingly gritty.

Microscopically the tumour was composed of rather straight interlacing bands of slender fusiform cells and dense strands of collagen. There were only a few, widely scattered whorls that were characteristic of meningioma. There were extensive regions of degeneration containing heavy deposits of calcium in the form of fine particles and larger concretions not related to whorls nor resembling the usual psammoma bodies.

Although the weight of the tumour equalled that of a normal brain, its smaller size was due to greater density and the high content of calcium (Fig. 2).

Postmortem Examination. There were discrete ulcers of the oesophagus, cardia of stomach and duodenum, and a perforation of the oesophagus, with extravasation of gastric contents into the peritoneal and both pleural cavities. This was apparently a terminal event, because there was no evidence of mediastinitis or peritonitis.

The cerebral convolutions were moderately flattened. The operative defect in the posterior part of the left hemisphere involved a large portion of occipital and parietal lobes and com-

municated directly with the body of the lateral ventricle. The lateral and 3rd ventricles were greatly enlarged (Fig. 3).

Both hemispheres were covered by fibrous subdural membranes, 0.2 to 0.3 mm. thick, separated by a layer of xanthochromic fluid. The outer membrane was adherent to the dura and the inner in contact with the arachnoid, but not adherent to it. This inner membrane dipped into the operative defect, continuing as an unbroken lining of the entire ventricular system. An outpocketing, about 2 cm. in diameter, extended through the tentorial notch, dorsal to the quadrigeminal plate. This and the underlying aqueduct were somewhat compressed and displaced to the right (Fig. 3, insert). Aside from this there was no evident obstruction to the outflow of CSF, as the foramina of Magendie and Luschka were patent.

Microscopic examination of the dura was not remarkable. The membranes were composed of dense, vascular, fibroblastic tissue with a slight infiltration of lymphocytes and polymorphonuclear leucocytes. There were a few deposits of blood pigment in the space between

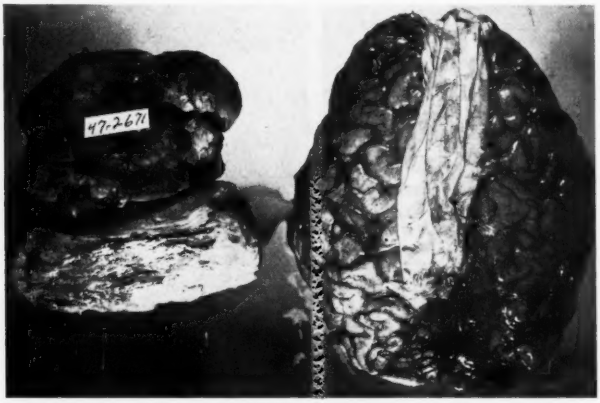

FIG. 2. Size of 1353-gram meningioma compared with a normal adult brain. In the specimen at the left the upper portion represents the 833-gram intracranial extension, the lower portion the 520-gram extracranial extension of the tumour removed at the first stage.

the membranes, but no exudate. On the surface of the brain at the site of operation there were recent haemorrhages, collections of phagocytes filled with blood pigment, and areas of exudate and lymphocytic infiltration. The ependyma of the ventricles was partly destroyed and replaced by a dense layer of fibrous tissue with underlying glial proliferation. This layer was 0.6 mm. in thickness, its surface coated with an exudate of neutrophilic leucocytes and beneath, in the subependymal zone, underlain by proliferating adventitial connective tissue and perivascular infiltrations of lymphocytes.

The protein-rich fluid surrounded by a membrane resembled a subdural hygroma. We have observed a very similar hygroma at autopsy after an operation for obstructive hydrocephalus. This communicated with the 3rd ventricle through a ventriculostomy opening in the lamina terminalis. We believe that the presence of large quantities of bloody CSF in the subdural space after operation led to the formation of membranes with encapsulation, as is the case in the development of subdural haematomas.

DISCUSSION

This case is reported not only because the tumour was so much larger than any meningioma of previous record, but because of several other unusual features. These include:

Time of Onset. Meningiomas rarely occur in childhood. In this case signs of increased intracranial pressure first developed in a child of 5½ years. In the series of 313 meningiomata reported by Cushing and Eisenhardt only 6 were found in preadolescents, and the youngest arising within the skull occurred in a child of 8.

Production of Cerebellar Signs by Compression of the Tentorium from Above. Nystagmus, clumsiness suggesting adiadokokinesis, and unsteadiness of gait at the time of the patient's first admission led to a tentative diagnosis of tumour beneath the tentorium. In Pilcher's case, where a 374-gram meningioma occupied the occipitoparietal region, the patient also had nystagmus and unsteadiness of gait, which

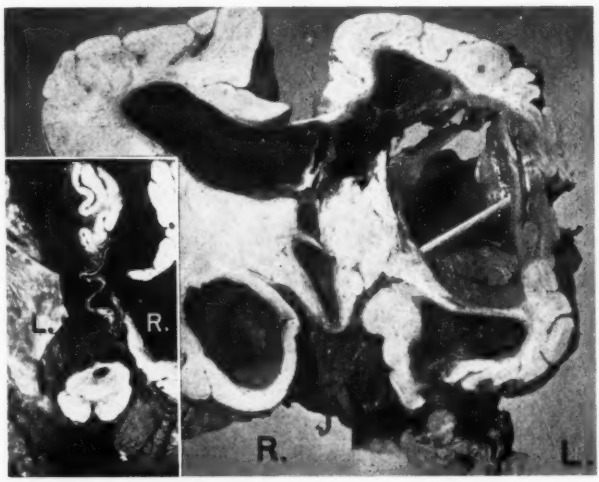

FIG. 3. Cross-section of brain through the thalamus, viewed from in front, showing degree of hydrocephalus and thick ependymal lining, which extended throughout the ventricular system. The larger left lateral ventricle can be seen opening into the operative cavity. *Insert:* The opposite surface of the same cross-section illustrates the extension of the hygroma through the tentorial notch with compression of the quadrigeminal plate and Sylvian aqueduct. R = right side, L = left.

together with deafness on the side of the growth raised the question of an acoustic tumour. Negative posterior fossa exploration in each case was averted by ventriculography.

Late Complications Following Removal of Tumour. The extensive subdural hygroma which filled the postoperative defect appears to have progressively interfered with the circulation of CSF by compression of the quadrigeminal plate and Sylvian aqueduct. As these membranes communicated with the ventricular system, fluid containing erythrocytes, white cells, and a rising concentration of protein accumulated within the cavity and ventricles in increasing amounts. As suggested by Finlayson and Penfield[3] and recently confirmed by Jackson[4] in animal experiments, the discharge of decomposition products of red blood cells in the subarachnoid space and ventricles may produce an aseptic meningitis and ventriculitis. These findings were features of unusual interest in the terminal illness 6 months after successful surgical removal of the tumour.

The tragic features in this case were the misleading biopsy at the primary operation and the prolonged refusal on the part of the parents to permit reexploration. If this could have been carried out within the first 6 years, before the onset of hemiparesis and convulsions, the patient might have made a satisfactory recovery with only a moderate reduction in visual acuity and a right homonymous hemianopsia.

SUMMARY

A meningioma of over 10 years' growth is reported with a record weight of 1353 grams.

Removal of the 520-gram extracranial herniation and secondary resection of the 833-gram intracranial mass from the parieto-occipital portion of the brain were accomplished successfully, but too late to prevent deterioration from cerebral atrophy and convulsive seizures. The boy died 6 months later of progressive hydrocephalus. An unusual subdural hygroma, which filled the postoperative cavity and compressed the Sylvian aqueduct, communicated with the ventricles.

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DUANE'S RETRACTION SYNDROME

A CASE REPORT*

John W. Chambers, M.D.

Veterans Administration Hospital, Perry Point, Maryland

(Received for publication February 16, 1950)

This case exemplifies a particular congenital anomaly[1,2] involving extraocular movements and is reported because of the great confusion in diagnosis which could have been avoided by careful observation, despite the misleading history. Errors in diagnosis in ocular anomalies are frequent as a result of failure to realize that congenital anomalies of the eyes are not uncommon and are often unnoticed by the individual patient—i.e. congenital Horner's syndrome, hyaline bodies of the nerve (Drusen) suggesting choked disks, tilting of the optic disks with bitemporal field defects suggesting a chiasmal lesion, pseudopapilloedema, pseudoneuritis and so forth.

REPORT OF CASE

W.E.S. (P.P.V.H. #20784), a white male aged 19 years, first complained in the fall of 1947, while in the army, of double vision on looking to the left and of inability to move either

* Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion of the Veterans Administration.

eye past the midline, laterally. One day while shaving he noted that he was unable to see the right side of his face on looking in the mirror and was unable to look to the right. He had never noted any trouble prior to this time and had encountered no difficulty in his physical examination on induction into the army. In February and March 1948 he had episodes of severe headache of varying location, duration and intensity, associated on several occasions with amaurosis lasting about 20 minutes. His description of his headaches was quite typical of migraine. He was also bothered by nervousness and restlessness, and was hospitalized as a result of these complaints, rather than the eye difficulties. His mother and aunt said that he had never been able to move his eyes to either side since birth.

Physical examination was negative except as regards the eyes. X-rays of skull showed no abnormality. CSF, blood and urine were normal.

Ophthalmological Consultation. "Paralysis of left eye on looking to left, on looking down and to left; paralysis of both right and left eyes on looking to right; paralysis of right eye on looking down and to right. Red lens diplopia test at 1 m. shows diplopia in primary position and in all cardinal directions of gaze.

"Diagnosis: Ophthalmoplegia, externa, O.U. with paralysis of rectus lat. O.D., rectus inf. O.D., rectus lat. O.S., rectus inf. O.S., rectus med. O.S.

"Findings suggest a lesion involving tissues in area of cavernous sinus, bilateral. The possibility of a basilar arachnoiditis on a luetic basis should be considered."

Neurological Consultation. "Vision unimpaired. Pupils normal. Eye-grounds show no lesions. No movement in either horizontal direction of left eye although upward and downward movement is practically normal. The right eye moves outward but not inward. Convergence is abolished. Diplopia is present as well as strabismus.

"Impression: History of headache, temporary blindness, diplopia, and lack of movement of eyes indicate an intramedullary lesion, although they could be due to basilar meningitis. ? Encephalitis ? tumor in aqueductal region. Air studies should be considered."

Other diagnoses included bizarre neoplasm and multiple sclerosis. After much difference of opinion patient was discharged from the army with a diagnosis of schizophrenic reaction and bilateral abducens paralysis, probably due to birth injury.

Admission to Perry Point Veterans Hospital, Mar. 19, 1949, for follow-up examination. Physical findings were normal except as regards the eyes. On lumbar puncture normal dynamics and pressures were noted. CSF was normal.

Ophthalmological Consultation. R.E.V., L.E.V. 20/20 (uncorrected). Fundi normal. Fields normal to form and color. Right pupil larger than left. Pupils react to light and accommodation. Paresis of right external rectus, left external rectus, left sup. oblique and left inf. oblique.

Impression: Nuclear lesion, because bilateral, with only partial involvement of 3rd nerve, with normal pupillary reaction.

Medical Consultation. ? Myasthenia gravis; 0.5 mg. prostigmine was given without benefit. Patient was then referred to neurosurgery.

Examination. There was inability to abduct either eye (Figs. 1, 2 and 3). On the left, adduction was poorly performed with *marked narrowing of the lid slit and retraction of the globe* when adduction was attempted. Adduction was slightly limited on the right with slight narrowing of the lid slit and barely noticeable retraction of the globe.

Impression: Bilateral retraction syndrome (Duane's syndrome).

Comment. This condition occurs more often in females, with the left eye more frequently affected than the right. It may be familial. On pathological examination there is more or less complete absence of muscular tissue with many inelastic fibrous bands. Most observers feel that injury does not play a role. The *syndrome is not associated with any abnormality of the central nervous system.* There is limitation of abduction, usually complete. Sometimes there is fuller movement in the upper and lower fields as result of abducting power of the oblique muscles. Retraction of the globe occurs on attempted abduction (1–10 mm.) and narrowing of the palpebral

fissure occurs. There may be limitation of adduction. The retraction is generally explained on the basis of lack of stretch of the fibrous external rectus which steadies the globe when adduction is attempted, while the other recti muscles exert their influence as retractors. The narrowing of the palpebral fissure is most readily explained on the basis of retraction of the globe. There are no associated pupillary or visual disturbances. Subjective symptoms are usually conspicuous by their absence.[3,4]

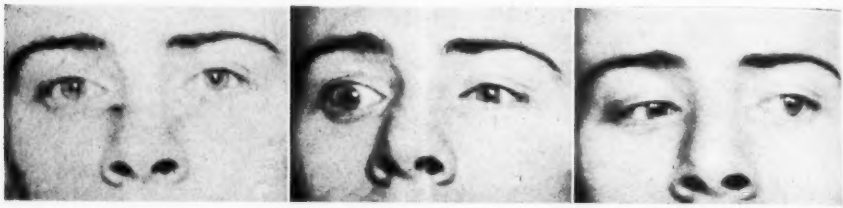

FIG. 1. Eyes straight.

FIG. 2. Attempted right lateral gaze.

FIG. 3. Attempted left lateral gaze.

The abduction defect and the retraction with narrowing of the lid slit can be clearly seen in the above figures.

SUMMARY

A typical case of the retraction syndrome is presented for the purpose of indicating the diagnostic confusion that may occur and of pointing out the typical features that enable a certain diagnosis to be made. It is hoped that cases falling into this category will not in the future be so frequently misinterpreted as 6th nerve palsies or other intracranial lesions.

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AN IMPROVED MODEL OF A HUMAN STEREOTAXIC INSTRUMENT

ROBERT HAYNE, M.D., AND RUSSELL MEYERS, M.D.
Division of Neurosurgery, College of Medicine, State University of Iowa, Iowa City

(Received for publication February 11, 1950)

A stereotaxic instrument applicable to experimentation on the cat brain was first described in 1908 by Horsley and Clarke.[4] Twelve years later a similar instrument was developed by Clarke and Henderson[2] for use on *Macacus rhesus*. At that time, Clarke[1] envisioned the construction of a stereotaxic instrument for the human brain. However, it was not until 1947 that the development of such was announced by Spiegel, Wycis, Marks and Lee.[7] Priority for the development and first use of a human stereotaxic instrument belongs to these investigators.

In addition to the apparatus to be described below, at least three stereotaxic models have been projected—one by William Sweet, another by Jasper and Hunter[5] and a third by Talairach *et al.*[8]

The first model of the instrument that constitutes the basis of this report was designed by one of us (R.H.) in 1947 while he was a Fellow at the Illinois Neuropsychiatric Institute. It was built with the aid of a monetary grant from the Miller Fund and was employed by Hayne, Belinson and Gibbs[3] for the placement of pick-up electrodes in the thalamus of epileptic subjects.

At that time dependable human coordinates had not been established as a preliminary to placing the electrodes. This fact together with certain shortcomings of the instrument itself—particularly the uncertain manner of its fixation to the scalp—necessitated verifying the positions of the deep electrodes at the conclusion of each experiment by performing pneumoencephalography. The investigators became early impressed that even this method of verification was limited to studies of the gross structures lying in close apposition to the ventricular wall (e.g., the caudate head and medial portion of the thalamus). It did not appear possible without more reliable coordinates to infer with confidence the specific subcortical nuclei within which the various electrodes rested.

Through the courtesy of Dr. Frederick Gibbs, the model was transferred to the Division of Neurosurgery at the State University of Iowa where it was put through seven mechanical revisions and was subsequently employed on 26 cadavers for the establishment of three sets of coordinate systems applicable to (a) dolicho-, (b) meso- and (c) brachycephalic heads. It was exhibited in its revised form together with the coordinates in 1948.[6]

The present improved model incorporates five revisions beyond those developed in the first model. As with the latter, the present instrument is oriented on the skull in accord with the general principles employed by Horsley, Clarke and Henderson, i.e., with regard to three standard planes of reference: the *midsagittal*, the *interaural* and *Reid's base line*.* This orientation differs somewhat from the principle employed by Spiegel *et al.*[7] in which the pineal shadow constitutes a major point of reference.

The apparatus consists of two main parts—(i) *substructure* and (ii) *superstructure*. The substructure (Fig. 1) is essentially an orienting device. By means of its pointer-guides it is applied to the skull in such a manner as to relate it to three standard planes: the *midsagittal*, the *interaural* and the *horizontal plane corresponding to Reid's lines*. The substructure is immovably secured to the skull by introducing the paired pegs, FTr–FTl and Or–Ol, through stab-incisions in the locally

* Reid's line is an imaginary line connecting the inferior orbital rim of one side with the midpoint of the external auditory meatus of the same side.

anesthetized scalp and tapping them gently into convenient portions of the outer table of the fronto-temporal and squamo-occipital portions of the calvarium. The substructure is then ready to engage the superstructure. The superstructure (Fig. 2) constitutes a *carriage* by means of which the brain electrodes may be moved into any desired position on the tridimensional coordinates. The paired sleeves, Sr and Sl, of the superstructure are set down upon the engraved rods, Ghr and Ghl, and are in this manner engaged. The superstructure can be slid freely along these rods.

Fig. 3 illustrates the entire apparatus assembled and fixed to the calvarium so as to establish relations with the midsagittal (Mid-Sag.), interaural (Int-Aur.)

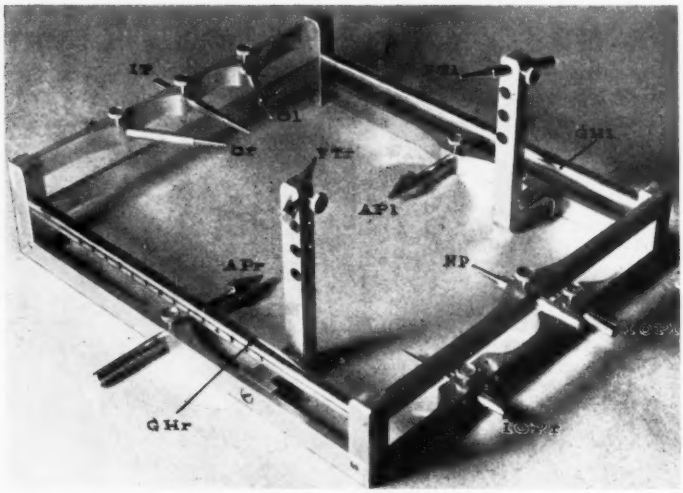

FIG. 1. Substructure seen from above. IOPr, IOPl = pointer-guides for inferior orbital rims, right and left; APr, APl = pointer-guides for external auditory meati, right and left; NP = pointer-guide for region of nasion; IP = pointer-guide for region of inion; FTr, FTl = fixating pegs for fronto-temporal regions, right and left; Or, Ol = fixating pegs for occipital regions, right and left; Ghr, Ghl = horizontal rods with engraved cm. scales on right and left sides of substructure. (The zero point of the scales corresponds to the interaural plane. Compare with Fig. 3.)

and para-Reid planes. Because the plane corresponding to Reid's line crosses the lowermost portions only of the human temporal lobe it does not constitute a convenient "zero" plane. For this reason the authors have arbitrarily adopted a horizontal plane 6.0 cm. superior to Reid's line and have designated it the para-Reid plane. The electrodes, Elh and Elv, may be advanced into and withdrawn from the brain by means of rack-and-pinion devices, RPv and RPt. The face of each rack exhibits a centimeter scale with millimeter subdivisions and a clamp, Cl, for securing the electrode shaft. The predetermination of the point(s) in the brain to be explored is, of course, dependent upon the coordinates.

The apparatus may be used for (a) mono- or unipolar stimulation, (b) electrocoagulation and (c) picking up electrical potentials from subcortical structures. The authors are at present employing it for further refinement of coordinates and for electrical potential studies.

This model weighs 1.87 Kg. (approx. 4 lbs., 2 oz.). All adjustments are accurate to 0.1 mm. and are well within the limits of experimental error.* The apparatus can be autoclaved or immersed in any standard antiseptic solution routinely used for surgical instruments. It can be built for less than five hundred dollars.

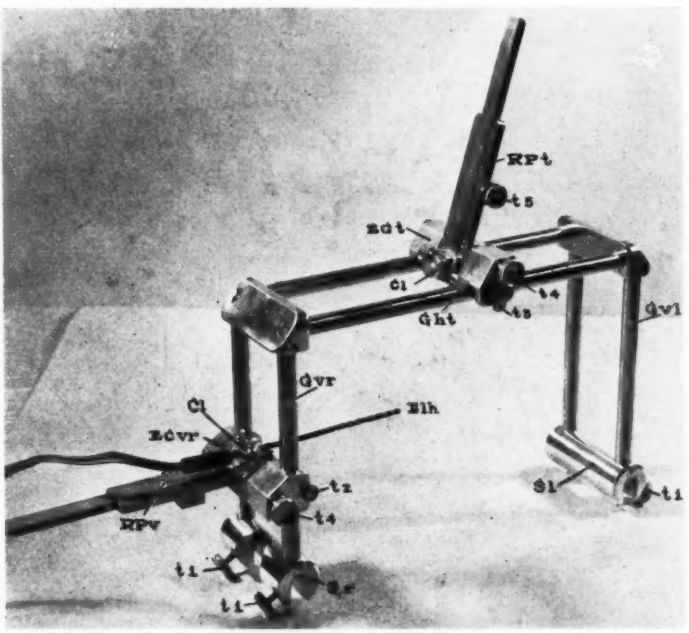

FIG. 2. Superstructure with its anterior aspect facing forward. Sr, Sl = sleeves for engaging the right and left sides of the substructure; t1, t1 = thumbscrews for fixing sleeves; Gvr, Gvl = anterior rods of the right and left vertical pairs, each of which carries an engraved cm. scale the zero point of which corresponds to the para-Reid plane; Ght = anterior rod of transverse horizontal pair carrying an engraved cm. scale the zero point of which corresponds to the midsagittal plane. ECvr = electrode carrier, right vertical; t2 = thumbscrew for fixing ECvr; ECt = electrode carrier, transverse; t3 = thumbscrew for fixing ECt; t4, t4 = thumbscrews for fixing electrode carriers in oblique positions; RPv, RPt = rack and pinion devices, vertical and transverse, for advancing and withdrawing electrodes; t5 = thumbscrew for operating rack and pinion devices; Cl, Cl = clamps for engaging electrode shafts; Elh = electrode shaft carrying 8 silver pick-up electrodes.

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* The writers desire to express their appreciation to Dr. Gerhardt von Bonin of the University of Illinois for valuable suggestions offered at the outset of this project; to Dr. W. Rex Ingram of the State University of Iowa for allowing use of the material in the anatomical laboratory; to Dr. Leonard P. Ristine, formerly Superintendent of the Mount Pleasant State Hospital, Iowa, and Dr. Emory Warner of the Department of Pathology of the State University of Iowa for their interest and cooperation. The present model was constructed by Mr. Joseph G. Sentinella of the technical staff of the Department of Physics, State University of Iowa, whose expert assistance is acknowledged.

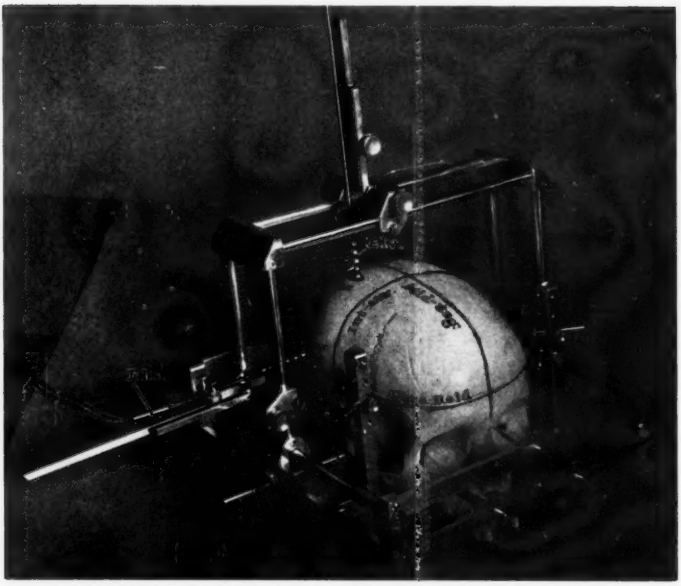

FIG. 3. Stereotaxic instrument assembled and fixed to the skull. Elh, Elv = vertical and transverse-horizontal electrode shafts; Br, Br = sites at which burr holes are indicated; Mid-Sag. = midsagittal plane; Int-Aur. = interaural plane; Para-Reid = para-Reid plane (6 cm. above Reid's lines).

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CAROTID-INTERNAL JUGULAR ANASTOMOSIS IN THE RHESUS MONKEY

ANGIOGRAPHIC AND GASOMETRIC STUDIES*

E. S. GURDJIAN, M.D., J. E. WEBSTER, M.D., AND F. A. MARTIN, M.D.

Department of Surgery, Wayne University College of Medicine, and Department of Neurosurgery, Grace Hospital, Detroit, Michigan

(Received for publication February 13, 1950)

AN ARTERIOVENOUS anastomosis between the carotid artery and the external jugular vein was performed by Gluck[3] in 1896. In 1902, Carrel and Morel[2] anastomosed the carotid artery to the external jugular vein in the dog. Later, in 1907, Watts[6] made pathological studies of the external jugular vein through which carotid arterial blood had traversed. Carrel suggested that such an anastomosis may "increase the circulation of the brain by directing a strong current of red blood through the internal jugular vein in dogs, the peripheral end of this vein being anastomosed through a central end of the common carotid artery." He quoted Jaboulay,[4] who in 1902 had expressed the opinion "that an arteriovenous anastomosis might have a good result in case of insufficient circulation, as in softening of the brain." Carrel did not believe that such a lesion could be successfully treated but stated that "it is not absolutely improbable that the slow and progressive disease of cerebro-sclerosis might be benefited by an operation modifying the circulation."

Carrel noted that when arterial blood was shunted into a vein, the vein wall rapidly thickened. The degree of thickening depended upon the amount of increase in the blood pressure. If the vein was required to support the normal arterial pressure, extensive thickening resulted with eventual complete obliteration of the lumen. Carrel found after the production of a shunt that "no clinical troubles occur in the dog; a dog operated on five months ago is living in good health." It is doubtful that arterial blood ever reached the brain in these animals even though the shunt may have been functioning. If the external jugular were used for such an anastomosis most of the blood would be shunted into the veins of the neck rather than into the cranial cavity.

Beck, McKhann, and Belnap[1] have reported upon common carotid-internal jugular anastomosis in the neck with ligation of the distal end of the internal jugular vein, as a means of revascularization of the human brain. A side-to-side anastomosis of the common carotid and internal jugular has been done and in later operations the external carotid and the internal jugular have been anastomosed. After the anastomosis an increase in the blood flow through the head was noted by utilizing a technique of tracing

* Aided by the Kresge Fund.

radioactive material injected into the circulation and gasometric studies of oxygen in the sagittal sinus before and after a shunt.

In order to evaluate critically carotid and internal jugular shunts, experiments have been performed on rhesus monkeys. Two fundamental factors have been studied and are discussed in this paper. (1) The direction of the flow of blood after the shunt. (2) The oxygen content of the blood in the sagittal sinus before and following the shunting procedure, studied by gasometric methods.

MATERIAL AND METHODS

The use of the dog as an experimental animal in this type of research is unsatisfactory since the internal jugular in this animal is small or absent and the external

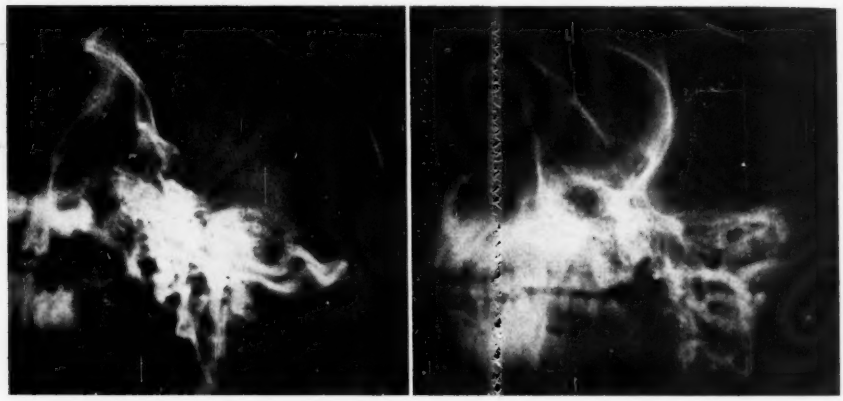

FIG. 1 (*left*). Angiogram of rhesus monkey after carotid-internal jugular shunt (5 cc. of 35 per cent diodrast injected). The diodrast goes up one jugular to the lateral sinus on the same side, thence to the opposite lateral sinus and down the other jugular in the neck. Note also that there are many other neck communications.

FIG. 2 (*right*). Angiogram of a shunted rhesus (5 cc. diodrast). Note that the internal jugular has extensive neck communications, although some of the diodrast reaches the intracranial space and enters the lateral sinus in the head.

jugular has extensive extracranial tributaries. The rhesus monkey, on the other hand, is suitable since the internal jugular vein and its communication with intracranial venous pathways is similar to that in the human (Figs. 1–5).

Thirty-eight animals were experimented upon. Intravenous Nembutal (1 gr./5 lbs. body weight) was used for anesthesia in all experiments.

In 11 animals angiograms were obtained after the shunt. Five cc. of 35 per cent solution of diodrast was used for visualization of the shunted pathways. Angiograms were obtained under varying conditions, including ligation of other veins on the neck such as the opposite jugular, external jugular and other neck tributaries; obstruction of both carotid arteries and ligation of the opposite lateral sinus. The pressure of the diodrast injection was measured in some experiments.

In 17 monkeys gasometric studies of oxygen content were made by the Van Slyke technique. Artificial respiration with intratracheal oxygen was used with 16

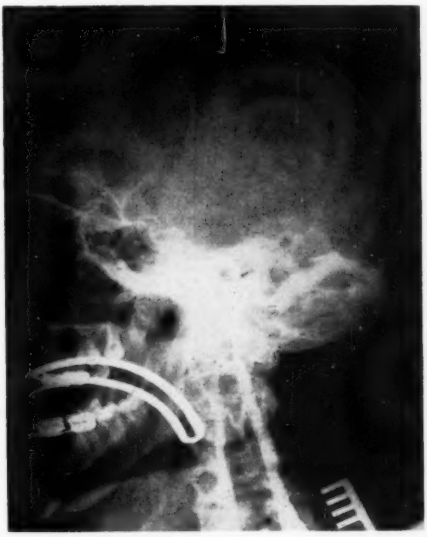

FIG. 3. A human left internal jugular venogram (20 cc. of 35 per cent diodrast injected by the open method). Note that the diodrast was directed up to the lateral sinus to the opposite lateral sinus and down the opposite jugular vein. There are intercommunications between the jugulars in the neck. The intracranial distribution of the diodrast is very similar to Fig. 1, a monkey angiogram.

strokes and 3 l. oxygen/min. During these procedures, which lasted between $2\frac{1}{2}$ to 4 hours, there was a decline in the oxygen tension in spite of artificial respiration with 100 per cent oxygen. However, in spite of this difficulty the figures are convincing (Table 1). In most animals a side-to-side anastomosis was used. In a few, an end-to-end shunt was utilized. Anastomoses were made on the right and left side in almost equal numbers. As shown in Table 1, specimens were obtained from the femoral artery, superior sagittal sinus, and the contralateral jugular before the shunt and after the shunt. Since these animals weighed from 8 to 10 lbs., it was not possible to obtain specimens from all of these vessels in the same animal, because of serious reduction of blood volume. However, in all animals the sagittal sinus blood oxygen content was studied before and after the shunt and arterial blood levels were established in most experiments and in others the oxygen content of the contralateral jugular before and after the shunt was obtained.

RESULTS

The angiographic studies revealed two patterns of flow of the shunted arterial blood. (1) The flow may reach the lateral sinus on the ipsilateral

TABLE 1

Oxygen tension in volume per cent of oxygen

Rhesus No.	Before Shunt Values			After Shunt Values		
	Sup. Sagittal Sinus	Contralateral Jugular	Femoral Artery	Sup. Sagittal Sinus	Contralateral Jugular	Femoral Artery
25	7.60	—	14.35	1.01	—	9.90
27	10.40	—	11.20	1.20	—	9.70
29	8.63	—	12.72	7.72	—	12.70
31	7.20	—	11.50	2.72	6.48	10.80
32	10.20	—	12.70	6.52	10.80	11.90
36	12.50	11.14	—	8.85	13.10	—
37	6.65	12.70	—	4.51	12.97	—
43	7.76	9.56	—	3.94	9.80	—

side, then course downward through the contralateral sinus into the neck veins. (2) The course may be upward in the jugular vein, to be dispersed through the basilar and neck tributaries (Figs. 1 and 2). The ligation of contralateral jugular vein or venous tributaries in the neck did not direct the flow into the intracranial venous system other than the lateral sinuses.

Angiograms obtained in shunted animals after ligation of the opposite lateral sinus revealed that the lateral, the superior sagittal, the inferior sagittal and tributaries of the cerebral cranial venous system were injected (Fig. 8). Such filling was possible only when injections of the diodrast in 5 to 10 cc. amount were administered with pressures well over 150 mm. of mercury (Fig. 6).

In Table 1, the gasometric studies of 8 animals are given. The oxygen content of the sagittal sinus blood decreased after the shunt was established. While the sagittal sinus values were quite low, the contralateral jugular vein oxygen content was indisputably high. The oxygenated blood became distributed in the neck veins. Ligation of the opposite lateral sinus did not result in an increase of oxygen content of the sagittal sinus blood. In this group of animals the sagittal sinus oxygen values were again uniformly decreased after the shunting (Rhesus No. 36, 37, Table 1).

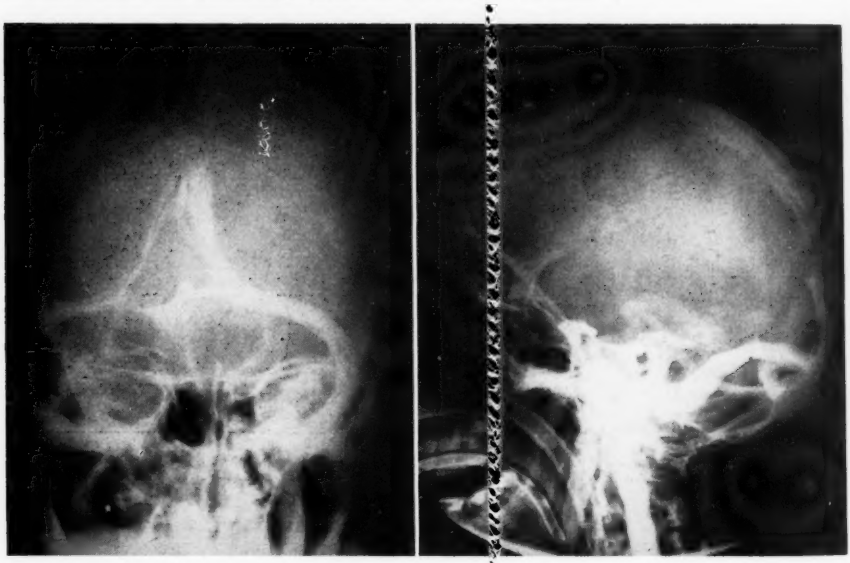

FIG. 4 (*left*). Injection of 20 cc. of 35 per cent diodrast by the open method into left internal jugular vein in the human. In the anteroposterior view the left jugular becomes continuous with the left lateral sinus and this in turn seems to be continuous with the sagittal sinus. This is different than the usual pattern of superior sagittal sinus being continuous with the right lateral sinus. Lateral view is shown in Fig. 5.

FIG. 5 (*right*). Injection of 25 cc. diodrast into left internal jugular vein. The diodrast traverses up the jugular into the lateral sinus on the one side to the opposite lateral sinus and then down the neck in the internal jugular on the opposite side. There are many neck communications. A portion of the diodrast is seen extending up toward the superior sagittal sinus without entering same.

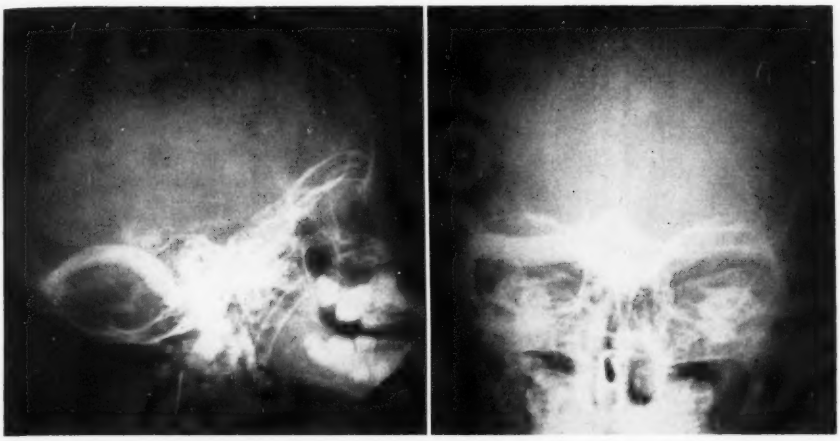

FIG. 6 (*left*). Angiogram after right common carotid-internal jugular fistula (20 cc. of 35 per cent diodrast). The dye goes up into the lateral sinus with no dye in the sagittal sinus (human).

FIG. 7 (*right*). Angiogram after right common carotid-internal jugular anastomosis (20 cc. of 35 per cent diodrast injected). The dye goes up the jugular to the lateral sinus on the same side, thence to the opposite lateral sinus. No dye is seen to enter the superior sagittal sinus. When the dye is injected into the common carotid artery immediately after completion of the shunt, the internal carotid and its branches, as well as the internal jugular and the lateral sinuses, are visualized.

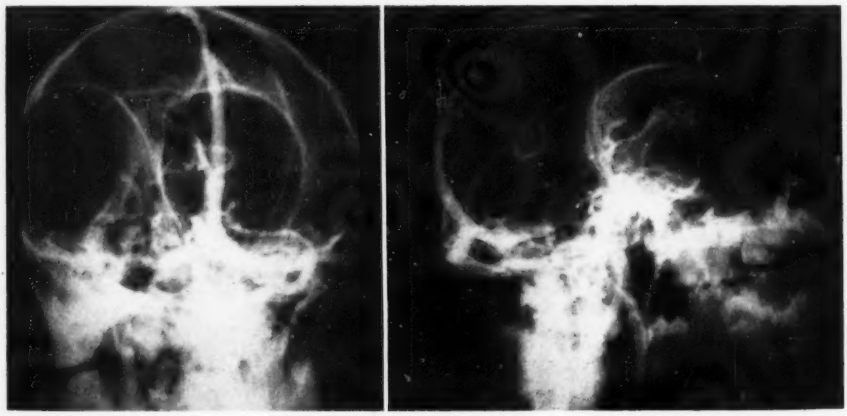

FIG. 8. Anteroposterior and lateral views of rhesus with right carotid-internal jugular fistula. The left lateral sinus was ligated. Note that the superior and inferior sagittal sinuses and cerebral tributaries are now visualized.

CONCLUSIONS

In acute experiments with the rhesus monkey after a carotid-internal jugular shunt, the blood does not flow into the sagittal, straight or petrosal sinuses for distribution by retrograde flow. Instead, it seeks a ready exit via the neck veins, the basilar veins and the lateral sinuses. Angiographic studies

show that the shunted flow can be directed into the intracerebral veins by ligation of the opposite lateral sinus and diodrast injection under pressure well above the systolic arterial pressure. As pointed out, among the animals with the opposite lateral sinus ligated, the oxygen volume per cent of the sagittal sinus blood did not become increased.

There appears to be a stagnation of the blood in the sagittal sinus after the shunting as shown by oxygen determinations in acute experiments. The lowered oxygen values in the sagittal sinus occurred in the presence of elevation of oxygen content of the neck veins, thus confirming the angiographic patterns.

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GENICULATE NEURALGIA

REPORT OF A CASE RELIEVED BY INTRACRANIAL SECTION OF THE NERVE OF WRISBERG*

ARCHER A. WILSON, M.D.

Neurosurgical Service, Charleston General Hospital, Charleston, West Virginia

(Received for publication February 18, 1950)

OUR teacher and friend, whom we are gathered here today to honor, has long been distinguished for his interest in the studies of the motor division of the 7th cranial nerve. It seems, therefore, particularly appropriate that your speaker, as one of his former students, should present a case involving the sensory division of this nerve.

It is my belief that the patient in this case has been correctly diagnosed as having geniculate neuralgia and that she has been relieved of her pain through intracranial section of the pars intermedia of Wrisberg. According to medical writing, this is one of the rarest of neurosurgical procedures. Because of the complexity of the sensory supply to the region of the ear and the rarity of the syndrome here presented, it might be of interest to review briefly some of the high lights in the evolution and development of our present knowledge of the functions of the 7th nerve.

Its anatomy as we know it today was described by Eustacius early in the 17th century. Sir Charles Bell[2] was the first to bring this nerve into clinical prominence by his studies on facial palsy.

For a long time the 7th nerve was regarded as purely motor in function From time to time an occasional sensory symptom has attracted attention, but such sensory manifestations have been referred to as produced by one of the many neighboring systems that converge and anastomose in this region.

In 1876 Webber[25] carefully described the pain phenomena observed in 6 cases of facial palsy. The pain was located in the ear and mastoid regions and in some cases, radiated to the face and occiput. He thought the pain was mediated through the trigeminal and the auricular branch of the vagus. Cushing[4] observed a crude sort of sensation persisting in the anterior two-thirds of the tongue, after posterior root section of the gasserian ganglion, in spite of anesthesia to touch, pain and temperature; later in one of his cases[5] a facial palsy supervened and this crude sensation then disappeared, indicating general sensory fibres in the chorda of facial origin.

Spiller[23] observed that light touch, pain and temperature sensations were lost in the trigeminal area after gasserian ganglion extirpation, but that deep pressure sensations were retained.

* Presented May 30, 1949 at a meeting of his former Residents, honoring Doctor Claude C. Coleman and commemorating the Thirtieth Anniversary of the establishment of the Department of Neurological Surgery, Medical College of Virginia, Richmond.

The investigations of His,[9] Retzius,[19] Sapolini[21] and others have conclusively shown that the facial nerve has a sensory ganglion, the geniculate; a sensory root, the pars intermedia of Wrisberg; as well as sensory fibres coursing in the chorda tympani, the great superficial petrosal nerve and the trunk proper of the 7th.

Ramsey Hunt[10] made his first report in 1907 on herpetic inflammations of the geniculate ganglion and for the next 30 years elaborated on the subject through numerous contributions,[11,12,13,14,15,16] which are responsible for our present knowledge of this highly complex system. Much of this knowledge was acquired through his study of herpes of the geniculate ganglion and he was thus able to map out the cutaneous zone of innervation of the geniculate ganglion into what is known as the "zoster zone," which includes the concha, tragus, antitragus, lobule and anthelix, the external auditory canal and a portion of the tympanum.

The pain in this condition is localized chiefly in the depths of the ear and in certain areas of the external ear, within the zoster zone. The pain is often lancinating and very severe and extends into the mastoid and occipital regions and into the face. These observations led to the recognition of the geniculate ganglion syndrome and established the fact that the facial nerve has a definite sensory system. Like the other mixed cranial nerves, the geniculate ganglion is composed of unipolar cells, the central processes of which terminate in the fasciculus solitarius of the medulla, with the central processes of the 9th and 10th, and constitute its sensory root. Hunt then summarizes his views on the geniculate system as follows:

1. Sensory filaments to the internal ear and branches to the zoster zone of the auricle, which bring it into close relation with the auditory mechanism. These branches are responsible for the otalgia.

2. The sensory system of the great superficial petrosal nerve, which brings the geniculate ganglion into close relation with the orbital, nasal and palatal branches of the sphenopalatine ganglion and the maxillary division of the fifth nerve. These branches are responsible for the deep prosopalgia.

3. A viscerosensory system subserving deep sensibility of the face, which is the seat of painful pressure sensations in geniculate prosopalgia.

These various branches of the sensory facial system have numerous anastomotic connections with the trigeminal, glossopharyngeal and vagal systems, as well as with the branches of the cervical plexus.

Not only are there these superficial connections, but the central sensory nucleus of the facial nerve stands in close anatomic relationship with those of the other mixed cranial nerves.

The central and peripheral associations with neighboring sensory systems account for the wide diffusion of pain in severe geniculate neuralgia.

It should be added that the facial nerve is especially rich in sympathetic and parasympathetic fibers, which explains the associated vasomotor and secretory manifestations.

Neuralgia facialis vera is characterized by pain in the distribution of the various divisions of the geniculate system. It may manifest itself as earache and as deep facial neuralgia. Geniculate prosopalgia is characterized by deep-seated pain in

the face in the posterior orbital, nasal, malar and palatal regions, associated with pressure pain sensations. There are associated otalgia and referred pains in the mastoid and the occipital region, at times extending to the neck and the shoulder.

In some cases geniculate otalgia predominates; in others deep prosopalgia is more in evidence. This is in harmony with neuralgia of the other mixed cranial nerves —the fifth, ninth and tenth—in which partial and incomplete forms are encountered.

I have used the term deep prosopalgia not only because it expresses the essential nature and localization of the pain but in order to place it in contrast with the prosopalgia of trigeminal origin, which is more superficial and with which it has been confused.

CASE REPORT

This patient had 5 hospital admissions under my care and 1 at a hospital in Detroit under neurosurgical care.

1st Admission. She was first admitted to the Charleston General Hospital, July 1, 1943, at the age of 46, having been referred by Doctor Burian of McVeigh, Kentucky.

She complained of pain in her right ear and cheek which had come on suddenly 5 weeks previously while she was waxing her floors. The pain appeared to be deep in the ear and radiated to the eye, lips and nose, and lower jaw. It had been constant and continuous since its onset and was not aggravated by talking, washing face, eating or swallowing.

Previous operations were appendectomy, cholecystectomy, sterilization and bilateral mastectomy for benign lesions of the breasts. She had had "sick headaches" for years, benefited by Gynergen.

She was a large woman who appeared to be in excellent physical health. B.P. 160/100 (on subsequent visits it was perfectly normal). There were no objective findings of significance. X-rays of skull, and blood, urine, and CSF studies were negative. She was discharged without diagnosis; it was felt that she did not have major trigeminal neuralgia.

2nd Admission, October 1943 (3 months later). She had the same complaints of pain deep in ear, radiating to face. Again routine studies revealed no abnormal findings and she was discharged, undiagnosed.

3rd Admission, March 1946 (almost 3 years after the 1st). Her complaints were the same and the pain had continued without interruption since its onset 3 years previously. She thought that at times it was made worse by washing hair and face, so the 2nd division of the right 5th nerve was avulsed. She stated that this procedure relieved her pain and she was discharged.

4th Admission (9 months later). She stated that she had remained completely relieved until 2 weeks before readmission, when she was awakened from a sound sleep by a sudden, sharp pain deep in her right ear. It seemed to spread out into her right face and eye, and over the nose and downward over the right neck. There had been no let-up in the pain, which she described as dull and aching in the ear "as though it is going to burst." She stated that the pain had been so "fierce" that she hadn't been able to retain much food. She felt that there was a trigger zone in front of the right ear.

Since she had gotten complete relief for 9 months through avulsion of the right 2nd division, it was felt that we were dealing with a major trigeminal neuralgia after all, and a selective retrogasserian neurectomy was carried out in October 1946. This

gave complete and immediate relief and on her 3rd postoperative day she ate a good lunch with the comment that "this is my first good meal in 4 weeks," and a few days later stated that she felt "grand."

She remained free of all pain and nothing further was heard from her till 13 months later.

4th Admission (Detroit). A telegram was received from her daughter in Detroit requesting information for the hosptial there. The patient had been suddenly seized with a violent deep right ear pain again and was hospitalized 10 days as a brain tumor suspect—but electro- and pneumoencephalography and other studies failed to reveal any evidence of a neoplasm. The opinion was expressed that she was suffering from a tension state with exaggeration of her symptoms.

She returned to her home in Kentucky and endured her pain with fortitude apparently for about a year, when there was an increase in intensity and a spread of her pain to the occipital region for the first time. During the next several weeks her husband spent, he stated, $50.00 for codeine, which she would use when her pain became unbearable. Many nights she was unable to sleep at all and would walk the floor crying out with her pain. It was in such a state that she reentered the hospital.

5th Admission, Mar. 13, 1949 (nearly 6 years after onset of pain). She had had no relief save for the 9 months following avulsion of 2nd division and 13 months following posterior root section of the 5th nerve.

She was quite obviously in very great anguish at this time. In addition to her deep ear and face pain she complained quite bitterly of tenderness in the occipital muscles, and the greater and lesser occipital nerves were infiltrated with a procaine ammonium sulfate solution without much relief. X-rays of cervical spine showed hypertrophic changes throughout.

After a week of observation and hours spent in talking with and examining her, the writer was firmly convinced of the genuineness of her complaints and of the necessity and desirability for a new approach to the problem, if relief was to be obtained. The clue was furnished by perusal of Ramsey Hunt's scholarly articles on geniculate neuralgia. Her symptoms appeared to be entirely in keeping with those of this malady.

On Mar. 22, 1949, operation was performed which she entered into with enthusiasm since it promised relief from her symptoms. Under local anesthesia a right unilateral subcerebellar craniotomy was done and the 7th, 8th, 9th and 10th cranial nerves were exposed. The 9th was touched first but because of the pre-operative Nembutal, her answers were not sufficiently clear-cut as to be dependable, so this nerve was severed. She stated that her old pain was still present and unchanged. The 8th nerve was then gently elevated and displaced backward with the shank of an angled hook knife, and the pars intermedia of Wrisberg picked up and sectioned. A definite answer that her pain had ceased was obtained. No bleeding was encountered and the dura and wound were closed.

A peripheral facial weakness developed postoperatively but since she still had corneal sensation, lid suture was not considered necessary. Otherwise her convalescence was uneventful. She left the hospital on her 9th postoperative day, entirely relieved of her former ear, face and neck pain. She reported for examination 6 weeks later and stated that she has had no suggestion of her former pain and her facial paralysis was clearing. She notes no especial change in her ear save that it is not quite as sensitive as the left though she does not recognize any numbness as such in the ear. Hearing was not impaired. She stated that during much of the time of her pain,

there was an excessive salivation so that she had considerable expectoration, nasal secretion and tearing and that all these symptoms have cleared since operation, save the tearing which was still present, presumably because of the facial paralysis.

A review of the literature reveals only 2 such patients operated on previously. The pain in both of the reported cases was paroxysmal rather than continuous.

The first case was reported by Clark and Taylor[3] in 1909 and the diagnosis was made possible by Hunt's earlier work. Their patient was a housewife, aged 28, who had had paroxysmal intermittent pain in front of the left ear for 2 years. About 8 months after onset, the pain had spread to involve the ear canal as well as the three branches of the 5th nerve and the occipital region. She was taking 12 gr. of morphine daily. Taylor severed the pars intermedia of the 7th nerve and the upper fasciculus of the 8th nerve with complete, immediate and permanent relief of pain with no further need or desire for opiates. This patient had a facial paralysis which was clearing 5 weeks later.

The second case report was that of Furlow[7] who operated on a young woman in November 1941 because of a shooting pain in the left ear of some 2 years' duration. Her pain was not continuous but would come and go spasmodically and was located chiefly deep in the ear, sometimes radiating to the face and neck. On one occasion cocainization of the sphenopalatine ganglion gave temporary relief. Apparently, this procedure interrupted a pain reflex as did the avulsion of the 2nd division and later, posterior root section, in our own case.

Reichert[18] in 1933 reported the case of a telephone operator, aged 31, with a similar history of a severe, intermittent, paroxysmal pain in the left ear. At times she was unable to use ear-phones because this disturbed the trigger zone on the concha and set off her pain. At times she also had aching pains in the left side of nose, face, eyeball, parieto-occipital and mastoid areas. He thought this was a case also of geniculate neuralgia, but upon operation under local anesthesia, her identical pain was reproduced when the 9th nerve was touched, so it was sectioned with complete and immediate relief. He had first gently touched the bundle of the 7th and 8th nerves and the patient complained of ear pain, but it did not have the characteristics of her old pain. This, then, was a case of tic douloureux of the branch of the glossopharyngeal (Jacobson's tympanic plexus branch). It is a variant of the glossopharyngeal neuralgia as it is typically found and illustrates again the complexity and overlapping in the mixed forms of otalgia in which the 5th, 7th, 9th and 10th cranial and the occipital nerves may play a part.

It is not surprising to have a partial neuralgia of a mixed cranial nerve. It is rather common to have a single division of the 5th nerve involved, later to spread to other divisions.

Since Reichert had thought he was dealing with a geniculate neuralgia and no doubt would have severed the nerve of Wrisberg had the patient been under general anesthesia, this illustrates the necessity of a local anesthetic

in such cases, as was first used by Davenport[1] in a case of glossopharyngeal neuralgia. It should be invaluable for the confirmation of clinical evidence in a case in which the diagnosis is in doubt. Had the pre-operative Nembutal not robbed our patient of her ability to cooperate accurately, her glossopharyngeal nerve probably would not have been sacrificed.

It is perhaps not surprising that the function of the sensory mechanism of the 7th nerve should so long have escaped detection. This is a small ganglionic system intercalated between the large system of the trigeminus in front and the systems of the glossopharyngeal and vagus and the upper cervical nerves behind. As a result, there are numerous anastomoses of which it is very difficult to determine their motor or sensory nature and their proper ganglionic origin. The problem is further complicated by the intricate relations of the facial system to the highly specialized structures of the auditory mechanism. That the distribution on the auricle was not recognized is also easily understood. The area is small and because of its situation in the auricular folds and the canal it is difficult of access. And further, the concentration of the auricular branches of the 5th, 9th, 10th and upper cervical nerves in the same region still more complicates matters, so that a small area of anesthesia in this region would rapidly fade in the overlapping of adjacent marginal areas.

In the matter of differential diagnosis, tic douloureux of the 5th and 9th nerves should cause no great difficulty as the symptomatology and treatment of these maladies is well understood and standardized. When one considers the great frequency of trigeminal neuralgia and the comparative frequency of glossopharyngeal and even of superior laryngeal (vagal) neuralgia, one should not expect an important sensory system like that of the geniculate to escape and no doubt many cases of this sort have masqueraded under the title of the atypical facial neuralgias.

A few years ago, a very distinguished visiting neurosurgeon, upon being asked to discuss atypical facial neuralgia, arose and stated simply: "I let them severely alone." He probably was being facetious but the problem isn't funny, either to the patient who has the malady or to the physician who assumes to treat such complaints and it behooves us all to take more seriously complaints of pain and make an effort to correctly diagnose them. Glaser,[8] analyzing 143 cases of atypical neuralgia, made a careful analysis of the symptomatology. He concluded that the pain was not superficial. It is not referred to the surface as in trigeminal neuralgia but is deep seated in the tissues, the bone, and the eyeball. The pain does not follow the distribution of the various branches of the 5th nerve but jumps anatomic boundaries and extends into the neck and arm.

In addition to pain, about half of the patients studied had associated sympathetic phenomena, which suggested the sympathetic system as the origin of the pain in atypical neuralgia. However, the late and to us here the much lamented Dr. Max Peet[17] concluded that atypical neuralgia could be due to sympathetic origin only through vasomotor spasm. Fay[6] spoke of a

syndrome of vascular pain to account for atypical neuralgia, expressing the opinion that pain follows the course of the vascular tree. In one case he performed 7 operations in an effort to cure a patient of atypical neuralgia: (1) Section of the trigeminal root; (2) resection of the sphenopalatine ganglion and the upper branches of the 7th nerve; (3) ablation of the cervical portion of the sympathetic trunk below the superior cervical ganglion; (4) stripping of the common carotid artery; (5) section of the hypoglossal nerve; (6) extracranial section of the glossopharyngeal nerve; (7) section of the sensory branches of the vagus nerve, the jugular branch of the vagus and Arnold's nerve and division of the sheath of the vagus and of vagus fibres to the internal and external carotid artery.

In Sluder's syndrome, Sluder[22] states "the pain of sphenopalatine neuralgia has a wide distribution and is localized in the root of the nose, in and about the eye and in the upper part of the face and the upper teeth and sometimes also in the lower jaw, the lower teeth, in the temple and about the zygoma, the ear, the mastoid, the occiput and the neck. It may extend to the shoulder and less often to the axilla and the breast, and in severe attacks, to the arm, the forearm, the hand and even the finger-tips." He also described vasomotor and secretory symptoms referable to the sympathetic system such as sneezing, congestion of mucous membranes with hydrorrhea, symptoms our patient had also.

Vail[24] stated that vidian neuralgia is characterized by pain in the nose, face, eye, ear and sometimes neck and shoulder.

Ruskin[20] concluded that the sphenopalatine ganglion syndrome of Sluder is "not a distinct clinical entity but is composed of four distinct syndromes: maxillary, sensory facial, sympathetic and local sphenopalatine (cells of the ganglion itself)".

Such symptoms as the above should certainly be carefully scanned as possible cases of geniculate neuralgia.

SUMMARY

A case is presented in which the symptoms appear to be entirely in keeping with what Ramsey Hunt has described as geniculate neuralgia, mediated through the sensory portion of the 7th cranial nerve. Section of the pars intermedia of Wrisberg has given complete and immediate relief of a severe and continuous pain deep in the ear, face, neck and occipital region of 6 years' duration, save for two periods of 9 and 13 months following avulsion of the 2nd division of the 5th nerve and posterior root section respectively. It is admitted that this statement is open to the criticism that not enough time has elapsed since operation to be sure of its permanency.*

And in closing, I would like to summarize the present status of neuralgic affections of the various cranial nerves in the words of Hunt.

1. True trigeminal neuralgia, which is distributed in one or more branches of the trifacial nerve and in which the pain is localized in the more superficial structures of

* Her complete relief continues in September 1950.

the face and intra-oral region. This is classic prosopalgia or trifacial neuralgia. In cases of neuralgia of the third division of the fifth nerve there is often associated otalgia.

2. Geniculate neuralgia, which involves the deeper structures of the face. This is characterized by pain in the deep posterior orbital, palatal and nasal regions, with painful pressure sensation in the face. This is geniculate deep prosopalgia, and with it there is associated geniculate otalgia.

3. Glossopharyngeal neuralgia, which is characterized by neuralgic pains in the distribution of the glossopharyngeal nerve at the base of the tongue and the adjacent regions of the throat and by associated otalgia.

4. Superior laryngeal neuralgia, of vagal origin, in which the pains are localized in the region of the larynx, with associated otalgia.

All these various forms are accessible to surgical intervention by the cranial method of approach through the posterior fossa, which exposes the fifth, seventh, ninth and tenth cranial nerves. If this procedure is carried out with the use of local anesthesia, it is possible by touching any one of the nerves to reproduce the neuralgic pain, thus confirming the clinical diagnosis, which is often involved and difficult.

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THE VAGUS NERVE IN SURGICAL CONSIDERATION OF GLOSSOPHARYNGEAL NEURALGIA*

JOHN T. ROBSON, M.D.,† AND JOHN BONICA, M.D.
Tacoma, Washington

(Received for publication February 20, 1950)

GLOSSOPHARYNGEAL neuralgia is a syndrome of tic-like pain at the angle of the jaw, with radiation frequently appearing at the external auditory canal. It may be confused with trigeminal neuralgia[1,3] of the 3rd division of the 5th cranial nerve and with neuralgia of the superior laryngeal nerve. Cocainization of the tonsillar area ordinarily serves to relieve and thus diagnose glossopharyngeal neuralgia, and cocainization of the pyriform fossa of the larynx is said to relieve neuralgia of the superior laryngeal nerve.[2]

Because of the anatomic and physiologic association of the 9th and 10th cranial nerves, it would not be surprising to find clinical association of the two in neuralgia at the angle of the jaw, but to our knowledge, this has not been reported. It is the purpose of this paper to report involvement of the vagus nerve in so-called glossopharyngeal neuralgia, and to report an improved method of diagnosis that we believe essential to the recognition of the unusual case where both the 9th and 10th nerves are involved in neuralgia and so to take appropriate surgical measures for relief.

CASE REPORTS

Case 1. V. J., a 43-year-old married female, was seen for the first time at the Tacoma General Hospital on Dec. 31, 1948. She gave a 5-year history of episodic pain radiating from the angle of the jaw to the left ear, "some way related to swallowing." General physical and neurologic examinations were negative. X-ray of the skull showed a "chronic left mastoiditis," although the patient was afebrile, and the left tympanic membrane showed no evidence of inflammation. Pain was relieved by cocainization of the left tonsillar fossa. Intracranial section of the left glossopharyngeal nerve was done Jan. 11, 1949, with relief of pain. Her course was uneventful and she was discharged on the 9th postoperative day.

Case 2. LeR. I., male, a 62-year-old retired school teacher, was seen Mar. 16, 1949. He gave a 7-year history of painful "spasms of the throat" initiated by talking and swallowing. Pain did not radiate to the ear. He had gone as long as 3 months without speaking because of fear of pain, and had been forced to give up his profession. Pain was relieved by cocainization of the left tonsillar fossa. He was admitted to the Tacoma General Hospital Mar. 16, 1949, where examination was negative except for electrocardiographic evidence of chronic myocardial anterior infarction, and the presence of a small rectal polyp. The left glossopharyngeal nerve

* Presented to the Staff Meeting of the Tacoma General Hospital, May 3, 1949.

† Medical Arts Building, Tacoma 2, Washington.

was resected intracranially on Mar. 24, 1949, and the patient was discharged on the 7th postoperative day, completely free of pain.

Case 3. A. E. K., a 62–year-old female, was seen Mar. 23, 1949 at the Tacoma General Hospital. She gave an 8-year history of pain in the left side of the pharynx, radiating to the ear. This was episodic in nature and initiated by swallowing. Cocainization of the left tonsillar fossa relieved the pharyngeal pain only. Pontocaine block at the jugular foramen resulted, on two occasions, in complete relief of pain. Examination was negative, as were skull x-rays and routine laboratory studies. On May 19, 1949, the left glossopharyngeal nerve was sectioned intracranially. This did not relieve pain in the left ear and on May 24, 1949, the wound was reopened and the anterior half of the vagus nerve sectioned. This resulted in complete relief of pain, and the patient was discharged on the 6th day after the 2nd operation.

Case 4. B. M., a 64-year-old female, was seen Dec. 28, 1949. She gave a 10-year history of pain in the right tonsillar area with recent development of pain in the right external ear. The pain was episodic and originally initiated by swallowing, but in recent years not so affected. Physical examination revealed evidence of gall bladder disease, and blood studies showed the prothrombin time consistently below 50 per cent of normal. Cocainization of the tonsillar fossa did not relieve pain. She was admitted to the Tacoma General Hospital Jan. 3, 1950, for study and correction of the prothrombin deficiency. Pontocaine block of the right jugular foramen resulted in complete relief of pain. On Feb. 9, 1950, the 9th and anterior half of the 10th cranial nerves were resected with complete relief of pain. Her course was uneventful and she was discharged the 6th day after operation.

COMMENT

Four cases of glossopharyngeal neuralgia are presented, and all the patients received complete surgical relief of pain. Cases 1 and 2 appeared to be cases of typical glossopharyngeal neuralgia. In Case 3, the patient was only partially relieved of pain by section of the 9th nerve, and incomplete section of the 10th nerve was necessary to achieve complete relief. This should have been anticipated because cocainization of the tonsillar fossa gave only partial relief of pain and block of the 9th and 10th nerves at the jugular foramen gave complete relief.

In Case 4 the history appeared to have been typical of glossopharyngeal neuralgia at the onset 8 years previously, but at the time the patient was seen, the pain was largely in the ear and was unrelieved by cocainization of the tonsillar fossa. Jugular foramen block, however, gave complete relief of pain, and surgical section of the 9th nerve combined with partial section of the 10th nerve gave immediate surgical relief of pain.

SUMMARY

Four cases of glossopharyngeal neuralgia are presented, in 2 of which the 10th nerve appeared to be involved. Cocainization of the tonsillar fossa, combined with pontocaine block at the jugular foramen, made it possible to recognize this condition before surgery, and institute appropriate surgical attack.

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EPIDURAL GRANULOMATA IN THE PRESENCE OF TANTALUM PLATES*

ARNOLD M. MEIROWSKY, M.D.,† LOUIS A. HAZOURI, M.D., AND D. JAMES GREINER, M.D.

Section on Neurological Surgery, Surgical Service, and Laboratory Service, Veterans Administration Medical Teaching Group, Kennedy Hospital, Memphis, Tennessee

(Received for publication February 20, 1950)

SINCE its introduction into neurosurgery,[2,8] tantalum has been widely used for the repair of cranial defects. The large number of penetrating wounds of the brain during the recent emergency has afforded an excellent opportunity to employ tantalum in a wide variety of instances. There have been a number of thorough reports in the literature, indicating the efficacy of tantalum. While the enthusiasm with which this apparently non-cytotoxic and fairly malleable metal has been received is no doubt well justified, there seems to be a need for emphasis on the complications which can readily be encountered in the use of tantalum plates for cranial repair. Bradford and Livingston[1] have enumerated these complications and have demonstrated failure in cranial repair with tantalum in 8 cases. These authors have already clearly stated the prerequisites for successful cranioplasty with tantalum.

It is the purpose of this communication to report an additional 11 cases in which removal of a tantalum plate was necessitated by the formation around the foreign body of a large infected epidural granuloma. Knowledge of this particular complication, which often does not manifest itself clinically for a long time after cranioplasty, seems particularly important in view of recent repeated advocation of the use of tantalum in the presence of infection.[4,9]

CASE REPORTS

Two of the following patients (Cases 1 and 2) were seen and operated on by one of us (AMM) at time of the original injury. Both these patients as well as the other 9 received a cranioplasty elsewhere and were not seen by us until the symptomatology of an epidural granuloma manifested itself.

Case 1. R. D. received a through-and-through bifrontal gunshot wound in June 1945, which resulted in severe cerebritis and meningitis. The destruction of both frontal sinuses exposed the patient to repeated secondary infections. A solid tantalum plate was used for cranioplasty, which was performed elsewhere 1 year after the injury. The presence of a continuous low-grade infection beneath the plate manifested itself by repeated attacks of conjunctivitis and one acute attack of iritis

* Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are a result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

† 3922 La Salle Court, Meadowbrook, Indianapolis, Indiana.

of the left eye resulting in blindness. Removal of the tantalum plate and resection of an extensive epidural granuloma resulted in cure.

Case 2. F. K. received a compound, comminuted, depressed skull fracture by blunt injury in July 1945. Successive insertion of 3 tantalum plates elsewhere within 3, 7 and 10 months after injury was performed in the presence of a persistent chronic extradural infection. Permanent removal of the tantalum plate and total resection of the extradural granuloma, which appeared irregular in shape, consistency and color, resulted in cure. There were hemorrhages spread through the granuloma, one of them measuring 3 cm. in diameter. Some areas within the granuloma appeared necrotic, others were grayish-white and bluish in color. This granuloma represented the severest of all foreign body reactions that we have seen in the presence of tantalum and was comparable only to the pathology seen experimentally in the presence of cytotoxic metals.[3] Follow-up studies of this patient have been carried out over a period of 30 months. He has remained asymptomatic.

Case 3. C. H. was struck by an automobile tire in his left frontal region in June 1948 and received a compound, comminuted, depressed fracture of the frontal bone. Four weeks later a CSF fistula developed which closed spontaneously in 10 days. A solid plate was inserted into a potentially infected wound $2\frac{1}{2}$ months after injury. Conservative therapy with antibiotics did not alter the course of events which led to cure only following removal of the tantalum plate, epidural granuloma, and superimposed abscess.

Case 4. C. B. sustained a shrapnel wound of the brain in April 1945. According to his statement, shrapnel penetrated the left occipital bone and lodged in the right parieto-occipital region, necessitating bilateral craniotomy. Two months after injury, a tantalum cranioplasty was performed. In July 1948 the patient first noticed swelling and tenderness over the site of cranioplasty. This was accompanied by several episodes of chills and fever. On admission here an encapsulated abscess was found overlying the tantalum plate. The abscess was evacuated and the entire capsule was dissected out. Beneath this abscess was a solid tantalum plate, measuring not more than 3×2 cm. Between tantalum plate and dura a thin but grossly infected epidural granuloma was found and totally resected. The patient made an uneventful recovery. It is particularly of note that the bony defect in this instance was so small that it did not necessitate cranioplasty.

Case 5. T. G. sustained a gunshot wound of the right frontal area in February 1945. Because of recurrent drainage of pus from the original site of injury, he was operated on 4 times during a period of 5 months. The 4th operation included cranioplasty with a solid tantalum plate. There was a direct connection with the frontal sinus. The patient was asymptomatic for a year, then was treated by intermittent incision and drainage until August 1947 when he was admitted to Kennedy. Removal of the tantalum plate revealed grossly infected granulomatous tissue covering the outer surface of the tantalum plate. The patient made an uneventful recovery. Again, it is particularly noted that the bony defect measured only 2.5×1.5 cm., and actually did not necessitate cranioplasty.

Case 6. T. H. sustained a shrapnel wound of the right parieto-occipital region in June, 1944. The first solid tantalum plate was inserted 3 weeks after the injury. From the available records it appears that debridement was performed at the same

time. Only 4 months later this plate had to be removed because of an underlying abscess. Simultaneously, a solid tantalum plate was reinserted. After 2 years, clinical symptoms, indicating the presence of an epidural granuloma, manifested themselves leading to admission to Kennedy. A freely movable solid tantalum plate and extensive infected epidural granuloma were removed, closure was performed, and the patient made an uneventful recovery.

Case 7. K. F. sustained a shrapnel wound of the right frontal area in September 1944. A period of 5 months elapsed between injury and cranioplasty. Seventeen months elapsed between cranioplasty and onset of symptoms indicating the presence of an epidural granuloma with overlying abscess. Removal of the plate and resection of the granuloma resulted in cure.

Case 8. E. J. sustained a shrapnel wound of the right frontal area in January 1945 with extension of the depressed fracture into the frontal sinus. A solid tantalum plate was inserted into a potentially infected area within 6 months after injury. Subsequent to cranioplasty the patient had intermittent attacks of conjunctivitis and swelling of the right upper lid. One of these attacks led to admission to Kennedy Hospital in January 1948. A typical epidural granuloma was found beneath the plate. Operative removal of the plate and granuloma resulted in cure. The size of the bony defect was approximately 2×3 cm., actually not necessitating cranioplasty.

Case 9. H. B. received a compound, depressed fracture of the right frontal bone in an automobile accident in September 1943. Cranioplasty was performed 15 months after the injury. Symptoms indicating the presence of an epidural granuloma first occurred 7 months after the plating. At that time, a purulent fistula developed which was treated by the local physician with intramuscular penicillin and wet packs for a period of 2 years. On admission to Kennedy in September 1947 the tantalum plate could be recognized in the depths of a draining sinus. Removal of the plate with resection of an extensive epidural granuloma and subsequent primary closure resulted in cure.

Case 10. W. S. received a compound injury in the right temporoparietal region in July 1944. A solid tantalum plate was inserted 6 months after the injury in spite of a persistent infection. Clinical signs to indicate the presence of an epidural granuloma did not manifest themselves until 2 years later when the patient was admitted to Kennedy. On operation here a smooth, brownish-green, dull membrane was found to cover the plate. The inner surface of the membrane showed a deep, brownish discoloration with interspersed roughened areas. The tantalum plate was noted to be elevated (Fig. 1) and the entire defect underneath the plate was filled with a thick, mucus-like mass. Parts of this mass were black and others were red, yellow, and gray. It measured 3 cm. in thickness and had the typical appearance of an epidural granuloma (Figs. 2 and 3). Primary closure following removal of the plate and resection of the granuloma resulted in cure.

Case 11. E. B. received a shrapnel wound of the right parietal bone in November 1944. Four months following the injury, a solid tantalum plate was inserted elsewhere. Three years intervened between the time of cranioplasty and onset of clinical signs and symptoms indicating the presence of an infected granuloma. Removal of the tantalum plate and resection of the granuloma in January 1948 at Kennedy resulted in cure.

FIG. 1. Anteroposterior view of skull showing elevation of the tantalum plate on the right side (Case 10).

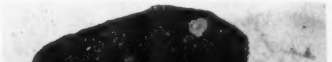

FIG. 2. Epidural granuloma (Case 10).

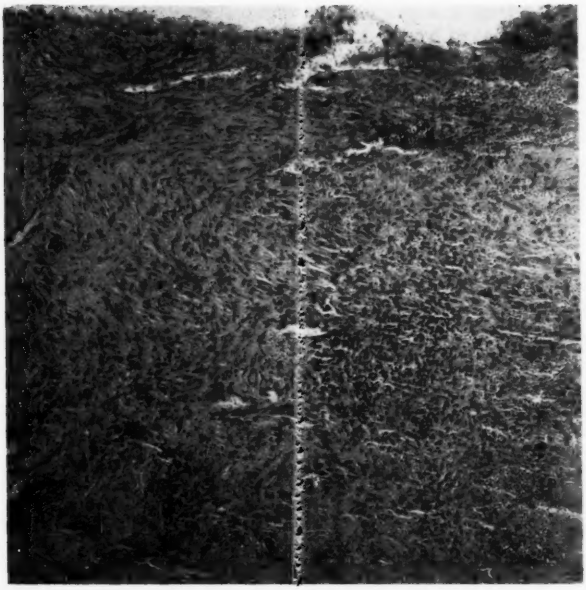

FIG. 3. Photomicrograph showing fibrous granulation tissue with small acute focus at right center (Case 10).

HISTOPATHOLOGY

The basic histopathologic changes are similar in each of the cases previously described. Chronic granulation is present directly in contact with the tantalum plate. This tissue is quite vascular, the vessels are of small calibre and many show some thickening of the vessel wall. There is an inflammatory cell infiltration composed chiefly of lymphocytes and plasma cells. Fibroblasts are prominent and these fade into a dense hyaline fibrous scar tissue in the outer portions of the sections. Vessels in these latter areas show sclerotic changes, chiefly marked intimal thickening. Lymphocytic perivascular infiltration extends from the areas of granulation into the scarred areas. Macrophages containing blood pigment are present in all cases both in the granulation and fibrosed tissue. This picture of chronic granulation and scarring shows a superimposed acute and subacute inflammatory reaction in some areas. There is a neutrophilic infiltration which varies in degree in the individual cases studied and which has progressed to necrosis of tissue in some instances. Bordering the acute reaction there is an infiltration by eosinophils and usually macrophages. There is marked congestion of capillary vessels and fresh hemorrhage is commonly present in the more acute areas. In the cases in which there is a draining sinus, the granulation tissue lining the tract shows both acute and chronic inflammation. These are the findings of a non-specific low-grade inflammation with attempted repair by scar tissue and with superimposed acute inflammatory reaction (Fig. 3).

STATISTICAL ANALYSIS

All 11 patients had suffered compound head injuries. Six of these injuries involved the frontal area, 1 the parietal, 1 the frontoparietal, 1 the parietotemporal, and 2 had penetrated the parieto-occipital lobes. In 4 patients, the penetrating wound of the brain communicated directly with the frontal sinus.

Shrapnel was the cause of injury in 5 cases; 2 patients suffered gunshot wounds. The remaining 4 were struck by blunt objects.

With the possible exception of Case 6, cranioplasty was not performed in any of these patients at the time of primary debridement. The time interval between injury and insertion of plate ranged from 3 weeks to 15 months. Nine cranioplasties were performed less than 6 months after the original injury. Two of the removed tantalum plates were found to have perforations; the remaining 9 were solid plates.

Onset of signs and symptoms indicating the development of an epidural granuloma subsequent to cranioplasty, ranged from 1 week to 33 months. In 8 of the 11 cases, symptoms developed 12 months after cranioplasty and later.

At time of admission to Kennedy Hospital, the skin had broken down over the plate in 8 patients. Purulent discharge with positive culture was found in all 8 patients. The remaining 3 patients presented themselves with fluctuant swelling, redness, and tenderness over the site of the plate.

At the time of removal of the plate, an infected granuloma (Figs. 2 and 3) was found in 10 of the 11 cases. In Case 5 an epidural abscess without fully developed granuloma, but with granulomatous tissue surrounding the plate, was encountered. In 4 cases the granuloma was covered by a frank abscess.

DISCUSSION

The most striking feature which applies to this entire group of cases is the fact that cranioplasty had been performed in the presence of actual or potential infection. The time interval between primary debridement and cranioplasty, although relatively short in the majority of instances, varied considerably in our cases. Rather than generalizing and setting a definite optimum time for cranioplasty there seems to be a need for return to the application of basic surgical principles in the indications for cranioplasty. Even though a metal may be non-cytotoxic, it is still a foreign body which is often poorly tolerated by tissues harboring infectious organisms. The cases reported by Bradford and Livingston,[1] Lane and Webster,[6] Lewin *et al.*,[7] as well as the cases reported in this communication, certainly suggest the inadvisability of repairing cranial defects with metallic foreign bodies in the presence of actual or potential infection.

In full agreement with Holbrook,[5] who has observed granuloma formation with acrylic plates in the presence of infection, we do not believe that the granulomata that we have described are specifically related to tantalum. It rather seems that these granulomata represent a general foreign body reaction of infected tissue.

The need for large perforations in tantalum plates is suggested by the fact that in 9 of the 11 cases the plates were solid. Granuloma formation does not seem to occur as readily in the presence of perforated plates and can certainly be recognized more easily.

The delay of clinical manifestations indicating the presence of a chronic epidural granuloma is particularly striking in the presence of solid plates.

It seems then that the indications for cranioplasty with tantalum should be guided by basic surgical principles. In the presence of actual infection cranioplasty is contraindicated. In the presence of potential infection, approximately 12 asymptomatic months should elapse before tantalum repair is attempted. Only large defects endangering the underlying structures should be plated.

In the presence of defects that communicate with the frontal sinus, only that part of the defect that does not cover the sinus should be plated. At no time should the tantalum be used to close an open sinus. The plastic rebuilding of orbital ridges and similar structures is dangerous and not warranted (Case 3). Light, perforated plates should be used. Cosmetic effects should give way to sound surgical principles and safety for the patient.

As soon as clinical manifestations of an epidural granuloma present themselves, surgical intervention, as advocated by Bradford and Livingston,[1] is mandatory. Removal of the plate and total excision of the granuloma, if

preceded by administration of antibiotics and chemotherapy, will prevent the threatening extension of the infectious process, and will usually result in cure.

SUMMARY

1. Eleven cases have been reported in which granuloma formation with or without overlying abscess necessitated removal of a tantalum plate.

2. The pathology, which is considered a general foreign body reaction of infected tissue rather than a specific response to tantalum, has been described.

3. In an attempt to prevent the dangerous complication of granuloma formation, the indications and contraindications for tantalum cranioplasty have been discussed.

4. The repair of cranial defects with tantalum plates in the presence of actual or potential infection is considered to be contraindicated.

We are indebted to Dr. Paul C. Bucy for his help and advice in the preparation of this study.

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THE USE OF PURE POLYETHYLENE PLATE FOR CRANIOPLASTY

EBEN ALEXANDER, JR., M.D., AND PETER H. DILLARD, M.D.
Department of Neurosurgery, The Bowman Gray School of Medicine and The North Carolina Baptist Hospital, Winston-Salem, North Carolina

(Received for publication February 20, 1950)

SKULL defects have been satisfactorily covered for many years with celluloid, tantalum, methyl methacrylate (Lucite, Plexiglass, Perspex), bone from other parts of the body, and various other substances, but each of these materials has one or more properties which make it disadvantageous for this procedure. Pure Polyethylene plate seems to have the desirable properties of all of the substances previously used without their disadvantages. It has undergone adequate experimental trial and has received limited clinical use. This synthetic plastic approaches the ideal so far as material for cranioplasty in human patients is concerned.

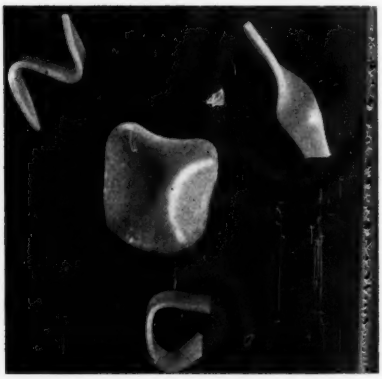

FIG. 1. Samples of Polyethylene plate, 3.25 mm. in thickness, molded in unusual shapes after immersion in hot water. The plate will retain these contours after it has been cooled.

Pure Polyethylene causes no immediate or delayed tissue reaction, and its physical properties are not apparently changed by contact with body fluid. It maintains contour and does not change its physical properties under the extremes of temperature variations to which the body is normally subjected; it is neither epileptogenic nor carcinogenic; it does not cast an opaque shadow in roentgenograms; and it is easily malleable when heated in boiling water to make it convenient for use at the operating table (Fig. 1).

Of the substances now in common use for cranioplasty, tantalum is the most popular among neurosurgeons.[5,8,16] It causes negligible tissue reaction and is easily molded at the operating table, but it is opaque to roentgen rays and, therefore, the air studies, angiograms, and other roentgenographic studies postoperatively are not usually satisfactory when tantalum has been used.

Methyl methacrylate[2,4,18,19] is not opaque to roentgen rays, but it is extremely hard at room temperature and requires a special technique for use at the operating table, which makes it cumbersome and inconvenient. It causes little if any tissue reaction.

Bone from other parts of the body has proved satisfactory in many hands

over the years. It is difficult to obtain contour in complicated bone defects with this substance, and its use requires two surgical incisions.

Celluloid[6,13] is easily molded to contour at the operating table and is not opaque to roentgen rays. It has received wide clinical use, but it is not a pure plastic substance and its physical properties change when it is buried in tissues. It becomes brittle in some cases and, like cellophane, causes an intense tissue reponse in both experimental animals and human subjects.

Other materials used for cranioplasty such as gold, silver, stainless steel, vitallium, ticonium and stainless steel wire mesh have one or more of the disadvantages mentioned above.

EXPERIMENTAL DATA

Pure Polyethylene in the form of tubing and film has been uniformly innocuous to tissue.[1,3,7,9,10] Certain forms of this material apparently contain-

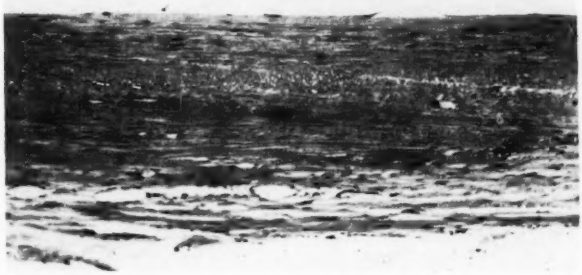

FIG. 2. (Dog. No. 73) Section of dura directly underlying Polyethylene plate 180 days after cranioplasty; there is no appreciable thickening.

ing impurities have been found by Poppe and de Oliveira[15] and by Yeager and Cowley[20] and others to cause intense tissue reaction and fibrosis, but in such instances the response is caused by impurities, not by the Polyethylene. Specifically, contact of Polyethylene with brain,[10] dura,[3] and bone[12] has been adequately studied and found to cause no significant tissue reaction. Since these experimental studies were made, Polyethylene in the form of plates varying from 2 mm. to 25 mm. in thickness has become available. In view of the rigidity of this material in plate form, it seemed to present itself as entirely suitable for cranioplasty. Laboratory investigation was, therefore, undertaken to determine the response of tissues in contact with Polyethylene plate and the ease with which it could be used in the operating room.

A total of 10 adult mongrel dogs was used to test this substance. The animals were operated upon under intravenous nembutal anesthesia, aseptic technique was used throughout, and skull defects measuring 2 to 4 cm. in size were created. These were then filled with pieces of Polyethylene plate,* 3.25 mm. in thickness cut to appropriate size, held in place with sutures of fine silk.

The animals were sacrificed at scheduled intervals from the 3rd to the 180th

* Obtained from Plastics Department, du Pont de Nemours & Co. (Inc.), Arlington, New Jersey. Now obtainable from Clay-Adams Co., Inc., New York.

postoperative day. After gross examination of the embedded plates and the tissues around them, specimens were taken of those tissues in contact with the plate, including the overlying temporalis muscle, the bone, dura, and the underlying brain. These were fixed in 10 per cent formalin solution, and embedded in paraffin. Sections stained with hematoxylin and eosin were examined microscopically.

There was in no instance any gross evidence of tissue reaction to the Polyethylene. In two instances, a layer of blood 2 mm. in thickness had collected over the surface of the plate, and in those cases the usual response to blood clot was observed. There was no alteration in the physical properties of the Polyethylene to gross examination.

Microscopic studies of the tissues showed no evidence of reaction. The

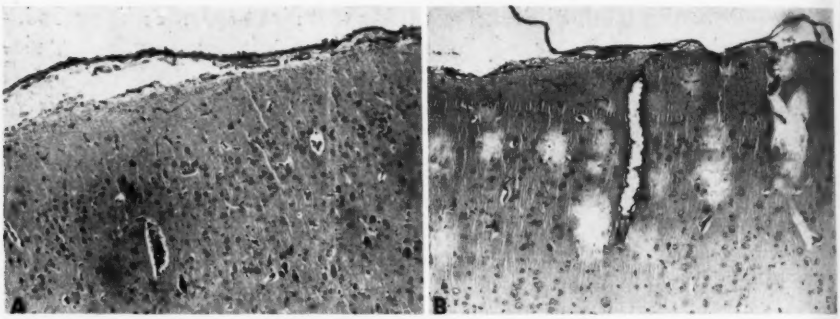

FIG. 3. (Dogs No. 73 and 90) Leptomeninges and cortex underlying cranioplasty site after 3 days (A) and after 180 days (B); there has been no immediate or delayed reaction to the Polyethylene.

dura was not significantly thickened (Fig. 2) and the underlying pia-arachnoid and brain showed no abnormality (Fig. 3). Beginning about the 10th postoperative day, a thin fibrous pseudo-membrane began to invest the plate and was present in all specimens examined after the 10th postoperative day. Figs. 2 and 3 show the type of response found in the tissues examined.

CLINICAL STUDIES

Polyethylene tubing and film have received extensive clinical trial,[1,3,7,11] and have been found to cause no significant tissue reaction in human patients. The physical properties of Polyethylene in these forms have not changed after months in the human body, and they have proved to be satisfactory for clinical use. Polyethylene plate has been used by Rubin, Robertson, and Shapiro[17] in reconstructive surgery of the face to replace bone and cartilage. In 1 case, a frontal skull defect 2 by 4 cm. in size was filled with Polyethylene plate "with excellent functional and cosmetic results."

Polyethylene plate has been used in 2 clinical cases to be described below. The technique employed will be discussed in some detail. The plate may be obtained in various thicknesses from 3 mm. to 8 mm., depending on the thickness of the bone surrounding the skull defect. The operative field

is prepared as for any cranioplasty, reflecting the scalp, removing the periosteum and scar tissue from the edges of the defect, and if necessary making a shelf through the outer table of the skull around the circumference of the defect into which the plate can be conveniently fitted.

Polyethylene plate is sterilized by soaking in 1:1000 aqueous solution of Zephiran for 18 hours, taking care to weight the plate, which has a low specific gravity, below the surface of the solution with a heavier object such as gauze. Care should be taken that the plate is grossly free of all foreign material before it is subjected to chemical sterilization and that all

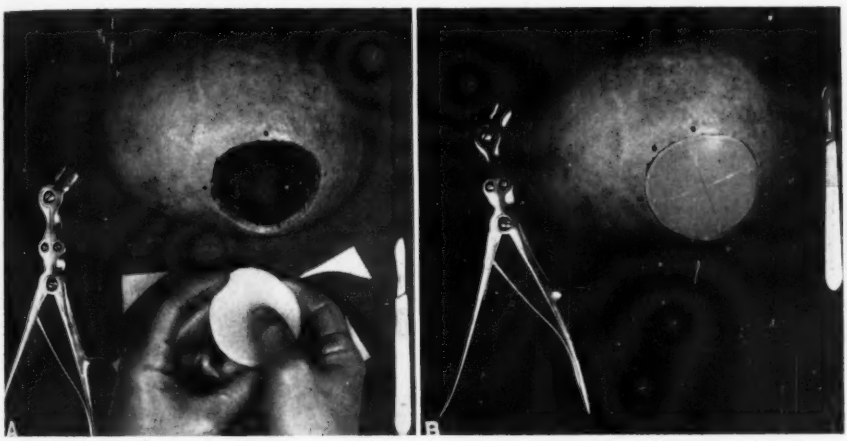

FIG. 4. (A) A piece of Polyethylene plate is being cut to fit the skull defect; it has been molded after immersion in boiling water. The scalpel and rongeurs are helpful in fashioning the plate. (B) The plate is in place. Tantalum wires are embedded in the surface for later roentgenographic identification.

surfaces of the plate are in contact with the solution during the entire period of sterilization. It may be boiled but will not withstand sterilization by autoclaving. The material is not affected at room temperatures by concentrated hydrochloric acid. Experiments have demonstrated the feasibility of sterilizing the material by preliminary immersion in concentrated hydrochloric acid followed by washing in sterile distilled water and further immersion in 1:1000 Zephiran until such time as it is needed at operation.

A portion of the plate is cut out with heavy scissors, knife, shears, or with the actual cautery which melts the plastic as it cuts through it, until it is of approximately the size of the skull defect. The edges can then be trimmed either with a scalpel or by the use of bone rongeurs until an exact fit is obtained (Fig. 4). Contour is easily established by immersing the entire plate in boiling water for 30 to 60 seconds, after which it can be bent or shaped as desired. Cooling the plastic quickly by immersing it in sterile cold water will serve to fix the contour established while the plate was hot. Minor alterations can be made by repeating the process until the plate conforms to the

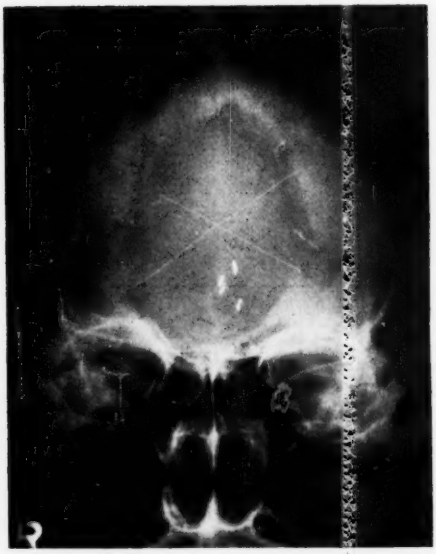

FIG. 5. (Case 2) Postoperative x-ray of skull. The thin tantalum wires embedded in the Polyethylene plate are visible although the plastic itself casts no shadow. Hemostatic silver clips are visible in the lower portion of the defect.

desired shape, even though it may be a complicated defect of the frontal bone. A plate can be made thicker in its central portion than at its edges to fill in the dead space of a large defect as necessary. Small drill holes are made in the plate and in the adjacent bone to fix the plate in place with silk or wire sutures.

Since the plate is not opaque to roentgen rays, it has been found helpful to embed fine tantalum wire in the superficial surface of the Polyethylene so that subsequent films will disclose the position of the plate. These wires are barely visible in postoperative films and do not obscure any detail of underlying structures (Fig. 5). Wire is conveniently embedded in Polyethylene by making grooves approximately 1 mm. in depth across the diameter of the plate with the point of the actual cautery, placing the wire in the groove, and sealing it in place by running the flat surface of the cautery over the surface.

Case 1. North Carolina Baptist Hospital Unit. No. 98563. W. D. F., an 8-year-old boy, suffered a compound comminuted depressed fracture of the left parietal region June 7, 1949. A skull defect, measuring approximately 6×8 cm., remained after debridement and closure of the wound. A cranioplasty, using Polyethylene plate, was performed on Sept. 15, 1949. A small sterile collection of yellow fluid was aspirated from the wound postoperatively, but otherwise the convalescence was uncomplicated and the result satisfactory.

Case 2. Kate Bitting Reynolds Memorial Hospital. S. W., a 37-year-old negro male, suffered a severe compound comminuted depressed fracture of the frontal bone on June 1, 1948. Debridement and closure of the wound 11 hours after injury were done at the Duke University Hospital. Purulent meningitis developed, which was successfully treated with penicillin.

The skull defect is shown in Fig. 6.

Cranioplasty with Polyethylene plate was done on Oct. 13, 1949. Fourteen days postoperatively, infection of the wound with hemolytic streptococcus and a paracolon bacillus was successfully cleared with local and systemic antibiotic therapy.

Four months after operation, he was doing well without evidence of infection or any reaction to the plastic plate (Fig. 7).

DISCUSSION

Polyethylene in pure form is innocuous to tissues and is inexpensive. In thicknesses of 3 mm. or more, it has adequate strength to maintain contour[14] and to offer protection against moderate injury. A piece of Polyethylene

FIG. 6. (Case 2) Frontal defect existing pre-operatively.

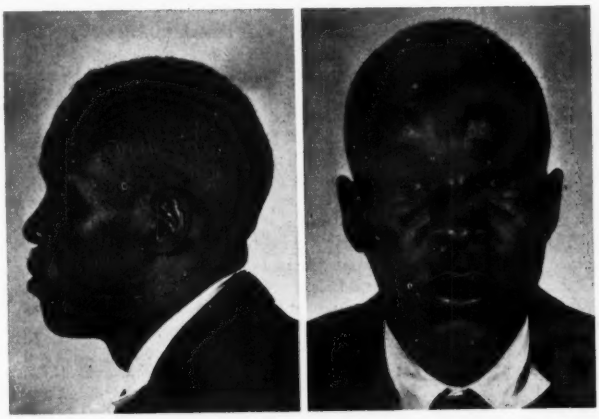

FIG. 7. (Case 2) Two months after Polyethylene cranioplasty.

plate 8 mm. in thickness bent to a 90° angle in boiling water and subsequently immersed in cold water cannot be returned to its original flat form by the ordinary strength of two hands working against the acute angle. This material not only offers adequate strength without brittleness, but it can be readily fashioned to fit complicated skull defects at the operating table without the use of special equipment or trained personnel necessary in the use of methyl methacrylate for cranioplasty. The continued use of this material is warranted, and the indications are that it will replace other materials, metal, plastics, and bone, now in use for cranioplasty.

SUMMARY

1. The advantages of Polyethylene plate in the performance of cranioplasty have been presented. It seems to possess all the qualities necessary for an ideal substitute for bone. It has the advantage of being radio-lucent, easy to fashion at the operating table, and of being innocuous to tissues.

2. An experimental study of this material has been presented.

3. Two clinical cases in which Polyethylene plate has been used for the repair of skull defects have been reviewed.

4. It is felt that further clinical trial will establish this plastic as the material of choice for cranioplasty.

Since the preparation of this paper, 3 cranioplasties using Polyethylene have been done with results similar to those described above.

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TRACTOTOMY FOR THE RELIEF OF TRIGEMINAL NEURALGIA

OBSERVATIONS IN 124 CASES

BENIAMINO GUIDETTI, M.D.

Neurosurgical Clinic, Serafimerlasarettet, Stockholm, Sweden*

(Received for publication February 25, 1950)

One of the chief objections to section of the 5th cranial nerve, according to Frazier, is the resulting total loss of sensation in the trigeminal area which, though tolerated by most patients, is always an unpleasant sequel. In some instances the operation has resulted in painful paresthesias in the anesthetic area.

To eliminate these complications Sjöqvist,[7] in 1937, as an outcome of his anatomical study of the trigeminal root, performed the first operative sectioning of the descending tract of the trigeminal nerve in the medulla oblongata, and obtained a dissociated anesthesia.

Since 1937 approximately 284 cases of tractotomy for various neuralgic syndromes have been reported from several different clinics. From the extensive material in Olivecrona's clinic I believe it is justifiable to draw conclusions regarding the indications for and against this method.

CLINICAL MATERIAL

During the years 1937 to 1948, 124 patients suffering from major trigeminal neuralgia have been operated upon at Serafimerlasarettet (in this series cases of bilateral neuralgia have not been included).

The patients might be divided into two groups, according to the level at which section of the trigeminal tract was performed: (Group 1) Those patients, a total of 40, in whom the tractotomy was performed at a level corresponding to the border between the middle and the inferior third of the olivary eminence, as originally recommended by Sjöqvist. These cases were reported on fully by Olivecrona[6] in 1942.

(Group 2) The 84 patients (1940–1948) in whom the tractotomy was performed at the level of the obex or, in a few cases, caudal to it. This modification of Sjöqvist's original technique was dictated by the conviction that most of the symptoms following tractotomy by his method were due to injury of the restiform body and the vagus rootlets. (Weinberger and Grant[3] are given credit for being the first to carry out this modified technique, but unfortunately their paper was not available in Sweden during the war.)

At the level of the obex, or a few mm. caudal, the trigeminal tract emerges from under the restiform body, and appears on the external surface of the medulla oblongata, forming at this point, between the posterior and lateral areas, a more or less pronounced elevation, called the tuberculum

* Chief: Professor H. Olivecrona.

cinereum. The tuberculum cinereum, as pointed out by Gonzalez,[1] is not always situated at the same level. Its position in the medulla oblongata varies not only in different individuals, but also in the two sides of the same individual. As a rule, however, it is pronounced a few mm. caudal to the level of the obex. Root section at this level, therefore, the spinal tract of the trigeminal nerve being no longer covered by the restiform body and the lowest rootlets of the vagus nerve having left the medulla rostral to this level, should not be followed by these complications.

OPERATIVE TECHNIQUE

The operative technique is the same as that described by Olivecrona in 1942.[6]

Even today a local anesthesia is preferred, because the pain experienced by the patient and the ensuing analgesia assist the surgeon to regulate the extent and depth of the incision.

The incision should be made at a level corresponding to the lower end of the 4th ventricle, or 1 to 2 mm. caudal to it, immediately lateral to the funiculus cuneatus, and extended ventrally to a point just dorsal to the level of the vagus rootlets. To ascertain the exact position of the sensory fibers of the 5th cranial nerve, it is a good plan to feel gently over the probable area with a blunt dissector. Location of the nerve will give pain in the corresponding side of the face. At the moment of incision, if it has been correctly placed, the patient will feel pain in the whole trigeminal area. If pain is felt only in the ophthalmic area, the incision should be extended a little in the dorsal direction. If pain is felt only in the mandibular area, the incision needs to be extended slightly in the ventral direction. To confirm that the incision has been correctly placed the sensation in the face is tested.

Great care must be taken to make the incision no deeper than 2 to 3 mm. in order to prevent damage to the ambiguus nucleus and sympathetic fibers. If the incision is extended too far in a dorsal direction the posterior columns and their nuclei may be damaged. If it is extended too far in a ventral direction, the spinothalamic and spinocerebellar (ventral) tract may be injured.

For neuralgia localized in the 3rd division, section of the trigeminal tract at the level of the obex does not always have the desired effect. If a cautious extension of the incision in a dorsal direction does not give complete analgesia, a new incision should be made slightly rostral to the first one.

OPERATIVE MORTALITY

In this series there have been 2 operative deaths (1.6 per cent). One patient died from pulmonary edema and a postoperative clot in the posterior fossa. The other patient died from a clot in the cerebellopontine cistern caused by bleeding from the tractotomy incision.

Autopsies were performed in each case.

RESULTS

Tractotomy was followed by loss or diminution of the sense of pain and temperature in the whole or part of the affected side of the face. The analgesia and thermoanesthesia were usually more marked during the early postoperative period than at a later stage, and as a rule went together. If there was complete thermoanesthesia, the patients were unable to discriminate between hot and cold water. Some patients had considerable impairment of their perception of pain, and others of their perception of heat, or cold, or both. These are not new or unexpected observations. Spiller long ago emphasized this dissociation, which he noticed in a case of thrombosis of the posterior inferior cerebellar artery, from which he concluded that there must be separate pathways for the perception of pain and temperature sensibility in the trigeminal tract.

The sense of touch was well preserved when tested with cotton wool or the finger tip. In some cases, however, there was slight subjective impairment, which corresponds to the observation made by Grant and his coworkers[2] that sometimes the number of touch points per square cm. was reduced and occasionally the threshold for touch points was increased. The majority of patients, however, noticed no diminution of facial sensibility after the tractotomy, except a slight difference between the two sides of the face when they ran their hands over the face, or when shaving.

Like practically all other writers on this subject, I found the ophthalmic division the most uniformly denervated, and the mandibular division the least denervated. In 85 patients, however, there was diminution or loss of pain and temperature sensibility in the mandibular division, which demonstrates that an analgesia of the 3rd division can be obtained more easily than is commonly thought possible. I shall refer to this again later on.

IMMEDIATE AND LATE NEUROLOGIC DISTURBANCES

Headache. In most instances the patients complained of severe headache for a few days after the operation, due undoubtedly to the loss of cerebrospinal fluid, and to penetration of air into the subarachnoid space.

Cerebellar Disturbances. During the first days following the operation, a high percentage (33 per cent) of the patients complained of cerebellar disturbances: incoordination of the arm and, less frequently, the leg on the side of the operation; weakness of the body on the affected side; disturbance of equilibrium with a tendency to fall on the side of operation; vertigo; and in some cases dysmetria of the arm, and nystagmus. Some of these patients, during the first weeks after operation, were obliged to use a support for walking.

As a rule these disturbances disappeared within a few weeks. Some patients found a certain difficulty in riding a bicycle, and walking in the dark, which persisted for some years. Only in 14 patients did slight disturbance in equilibrium persist for several months. (One patient, No. 25, earlier re-

ported on by Olivecrona,[6] and in whom the operation was complicated by hemorrhage causing a hematoma in the posterior fossa, was still unable to walk without support 5 years after operation.)

These complications are probably due to injury to the restiform body, because they usually occurred in cases in which section of the trigeminal tract was performed at a level rostral to the obex, at which level the trigeminal fibers are still covered by the restiform body. Out of the 40 patients in this series in whom the tractotomy was performed from 2 to 6 mm. rostral to the obex, 27 (67.5 per cent) complained of these disturbances. In 14 patients the disturbances disappeared within a few weeks, in 12 they persisted for several months, and in 1 for 5 years (the case mentioned earlier).

Of the 84 patients in whom the tractotomy was performed at a level corresponding to the lower end of the 4th ventricle, or caudal to it, these disturbances occurred in only 14 (17 per cent), in 13 disappearing within a few weeks, and in 1 persisting for several months.

According to Sjöqvist, these complications occur more frequently in older subjects than in younger. In our series I controlled this observation, and found the average age of patients who exhibited these disturbances to be 54, and of those who had no postoperative complications, 48.

Ataxia. Ataxia of the arm on the side of operation was observed in 3 patients. As it was connected with decrease of the sense of touch, and impairment of the sense of discrimination and position, it was believed that the nucleus of Burdach had been injured. This complication was noticed in a further 2 patients, but an accurate examination of the sensibilities could not be made.

Ataxia occurred more frequently in cases in which the incision had been made at the level of the obex, than in cases in which it had been made somewhat rostral. This complication is to be dreaded, especially when the neuralgia involves the 3rd division, in which case extending the incision too much in a dorsal direction can cause damage to the nucleus of Burdach.

Recurrent Nerve Paresis. This complication on the side of operation occurred in 8 patients; in 4 it was severe, and in 4 comparatively slight. In 6 of them the tractotomy was performed at the level rostral to the obex, in 1 at the level of the obex (slight paresis), and in 1 (severe paresis) at a level 2 mm. caudal.

In my opinion the most frequent cause of this complication is injury to the vagus rootlets at the level at which they pass through the spinal trigeminal tract. i.e., the middle of the olivary eminence. In the majority of patients with this complication the tractotomy had been performed at a level between the middle and inferior third of the olivary eminence. A too deep incision, injuring the nucleus ambiguus, may certainly be the cause of a recurrent nerve paresis, but, as the nucleus ambiguus runs through the length of the medulla oblongata, injury to a small group of its cells should not result in any lasting disability.[9]

Another mechanism which might be of importance, and probably was

the cause in the last case in the series, is the traction that might sometimes unwittingly be placed on the vagal roots at their point of exit from the medulla.

Horner's Syndrome. This complication was more or less complete in 6 patients, due to injury of the descending sympathetic fibers where they run in the reticulated substance, indicating that the incision had been made too deep. In most instances it disappeared within a short time, leaving no ill effects.

Corneal Complications. Although the corneal reflex was lost or diminished in the majority of the cases, slight neuroparalytic keratitis occurred in only 2, and was then due to the patients' own carelessness. This shows that the neuroparalytic keratitis, which is a frequent sequel of trigeminal tractotomy according to Frazier, is not caused by loss of the corneal reflex, but is most probably due to injury of the superficial petrosus nerve and the gasserian and perigasserian sympathetic fibers.

Lesions of the Spinothalamic Tract. In 10 patients the fibers of the spinothalamic tract were damaged, resulting in analgesia and thermoanesthesia of a more or less extensive area on the contralateral side of the body. This complication occurred more frequently in patients with neuralgia involving also, or exclusively, the ophthalmic area. This is due to the fact that the fibers of the 1st division run in the ventral part of the trigeminal tract, and an attempt to divide them can easily damage the spinothalamic tract, which at this level runs between the nuclei of the 5th cranial nerve and the inferior bulbaris oliva. In 7 of the patients the tractotomy was performed at the level of the obex, in 2 patients rostral to it, and in 1, caudal.

In 5 patients the postoperative analgesia and thermoanesthesia were limited to the foot and leg on the contralateral side; in 1 patient to the foot only; and in 1 patient reached up as far as the 12th dorsal segment. In 1 patient there was only analgesia in the contralateral foot and leg, while the temperature sensibility remained normal. In another it was noticed that whereas the analgesia reached to the 12th dorsal segment, the thermoanesthesia reached to the 3rd dorsal. Another case is worthy of mention:

Case 67-670/44. A man, aged 58, had had attacks of major trigeminal neuralgia in the 1st and 2nd divisions of the nerve for 2 years. Neurologic examination revealed nothing abnormal. As the neuralgia was principally in the 1st division of the 5th cranial nerve, it was decided to do a tractotomy. This was performed at the level of the obex. Tests after the operation showed that there were analgesia and thermoanesthesia within the region of the 1st and 2nd divisions, while in the area of the 3rd division all forms of sensation were normal. On the contralateral side of the body there was decided diminution in the sense of touch, pain and temperature as far as the 3rd cervical segment.

Three months after operation painful paresthesias developed in this area—excluding the face—which were described by the patient as a burning sensation. These paresthesias were not spontaneous, but were provoked usually by pressure on the affected side, as when sitting or getting into bed. They started as a burning pain, and spread over the entire half of the body up to the 3rd cervical segment,

without crossing over the midline. The same thing occurred if pressure was applied to the skin. Five years after operation these disturbances are still present.

On the whole, the loss of sensibilities and the slight paresthesias were well tolerated, and did not impair the working capacity, except in the aforementioned case, and in 2 others with slight paresthesias in the foot, on the contralateral side.

From these accidental lesions it might be argued, and rightly so, that the spinothalamic tract, at the level of the medulla oblongata, has the same arrangement as in the high cervical segments. The dorsal external fibers carry impulses from the caudal segments of the body; slightly deeper and medially, run the fibers carrying impulses from the lumbar and thoracic segments of the spinal cord; and centrally and ventrally, fibers carrying impulses from the cervical segment.

Spiller's observation that the sensibilities are not confused in the spinothalamic tract appears to be confirmed in the 2 cases mentioned earlier.

On the other hand, observations made in 1 patient with analgesia up to the 12th dorsal segment, and thermoanesthesia up to the 3rd dorsal, support the concept that the fibers carrying temperature sensibilities run in the posterior part of the spinothalamic tract. This agrees with the findings of Stookey[8] and others that chordotomy did not always abolish the temperature sensibility since its fibers lie in the more dorsal part of the tract.

Postoperative Paresthesias. Theoretically there is no reason why dysesthesia should not follow division of the descending tract of the trigeminal nerve, because it is well known that thrombosis of the posterior inferior cerebellar artery is frequently followed by severe painful paresthesias.

Eight patients complained of paresthesias, but in all 8 they were mild, not in any way comparable with the severe forms found after root section according to Frazier. Most of the patients felt "soreness" around the eyes, together with a slight burning sensation.

Recurrence. One of the most important points to be considered when evaluating the results of tractotomy, is the frequency of recurrence.

There were altogether 46 cases (37.1 per cent) of recurrence. Of the 27 patients in whom the recurring pain was severe, 14 had to be operated upon according to Frazier, and 1 according to Dandy. In 3 cases in which the first operation was a complete failure, with no analgesia and return of the pain, a secondary operation was performed immediately. The wound was reopened, and the incision into the medulla deepened. Two of these patients had recurrence of pain; in 1 it was slight, and in the other severe. Nine of the patients who had a severe recurrence of their pain did not wish to submit themselves to a further operation.

Among the remaining patients with recurrence, there were 5 in whom the recurring pain was mild, and 14 in whom it was slight. All of them can be said to have benefited from the operation.

It must be borne in mind that there is a risk of recurrence even after several years, depending on the degree of analgesia obtained. If complete

analgesia is not obtained in severe cases of neuralgia, the recurrent pain is likely to be as severe as the original pain, although sometimes (in 5 cases in this series) even complete analgesia of the trigeminal area is not a guarantee against recurrence.

In my opinion, from the point of view of recurrence, there is not much difference between those with mild, and those with severe forms of trigeminal neuralgia, though it might be thought that recurrence is more frequent in cases with severe neuralgia. Thirty patients in whom the trigeminal neuralgia had been severe, and 41 in whom it had been mild, had no recurrence of pain several years after the operation. Of the patients who had recurrence, 27 had had severe neuralgia, and 19 mild.

Furthermore, I did not find that the level at which the spinal trigeminal tract was severed, whether the obex, or caudal to it, in any way affected the recurrence.

In those patients with neuralgia involving also the mandibular area, the results of the operation as regards analgesia and recurrence were good in 20 patients, and poor in 10.

In those cases in which the neuralgia was localized exclusively in the 3rd trigeminal division, the operation gave good results in 5, and poor in 3.

In spite of these results, which do not really differ from those obtained in cases of trigeminal neuralgia localized in the 1st and 2nd division, I am of the opinion that for cases of neuralgia localized principally or exclusively in the mandibular area, tractotomy is not the method of choice. This is not because it is difficult to obtain analgesia in the 3rd trigeminal division, but because the operation is potentially more dangerous, the fibers of the 3rd division being situated in the dorsal part of the tract in the neighborhood of the dorsal nuclei. Furthermore, it is not always possible to get complete analgesia of the 3rd division by section of the trigeminal tract at the level of the obex. This would then necessitate section at a somewhat rostral level, resulting in unavoidable damage to the fibers of the restiform body.

DISCUSSION

To evaluate the therapeutic effect of any operation a long period of observation extended over many years is necessary, and a careful consideration of the risks involved compared with the advantages obtained.

Major trigeminal neuralgia is an extremely painful, but not fatal disease. To alleviate this condition, a surgical intervention is necessary which, with the lowest mortality rate, will relieve the pain without leaving disturbances that are more intolerable than the original pain.

Three surgical measures are used:

1. Subtemporal section of the sensory root (Frazier).
2. Suboccipital root section (Dandy).
3. Intramedullary section of the descending tract of the trigeminal root (Sjöqvist).

The transtemporal approach is very safe. In a series of 515 patients

operated upon in this clinic up to Dec. 31, 1948, the operative mortality was 0.77 per cent. This figure is very low when one considers the ages of these patients: 43 were over 75, 71 over 70, 87 over 65, 102 over 60. Among the 515 patients are included 25 operated upon for recurrence of pain. In the last 360 operations performed in this series, there was only 1 fatality.

Complications that may follow section of the 5th cranial nerve through the temporal fossa are facial weakness, keratitis, and paresthesias. Facial weakness and keratitis can, in the great majority of cases, be easily avoided. The only serious objection, therefore, to section of the trigeminal roots according to the Frazier method is the disability and discomfort caused by complete or nearly complete anesthesia in the greater part of the trigeminal area, and the paresthesias which sometimes occur in the same area.

Of the patients operated upon according to the Frazier method, the records show that 56 complained of slight paresthesias, and 16 of severe, continuous, burning paresthesias in the anesthetic area of the face. In these latter the former intermittent trigeminal pain disappeared after the operation, but was replaced by constant, intense paresthesias in the same anesthetic area, which were more distressing than the original spasmodic pain.

A preventive peripheric alcoholization, advised by many authors, to get patients used to the residual anesthesia, is not a guarantee against these complications. In some patients in whom a preventive alcoholization was done in other clinics without resulting in painful paresthesias, section of the sensory roots according to the Frazier method was followed by paresthesias in the anesthetic area of the face. These complications, at one time intractable, can today be overcome by the operation of lobotomy. (Of 3 patients in whom lobotomy was performed, 1 died after a few days from double pneumonia. In 1 patient the results were excellent, and in 1 they were good.)

The low rate of mortality, the infrequency of recurrence (theoretically there should be no recurrences after the temporal operation, but the 8.3 percentage of recurrences shows that the surgeon has been too conservative in a number of cases), the short period of hospitalization, and the insignificant number of complications, make section of the trigeminal sensory roots according to the Frazier method one of the surest and simplest methods of operation today. It is still the operation of choice for the majority of cases of major trigeminal neuralgia, particularly if the patient is old, or the neuralgia severe.[4]

Section of the sensory root at the pons according to Dandy's method was performed in most of the early cases in this clinic, but later only in cases with more exact indications.

A total of 145 patients were operated upon up to Dec. 31, 1948 by this method (in this series cases of bilateral neuralgia are not included.) Keratitis, facial weakness, and paresis of the masticatory muscles seldom occurred, and the records show that only 8 patients complained of very slight paresthesias in the anesthetic area of the face, due to the fact that with Dandy's method there is very insignificant, if any, loss of tactile sensation in the face.

In spite of these advantages, the method is unsuitable for routine operations, because of the high mortality rate (3.4 per cent), and the greater frequency of recurrence (17.9 per cent) compared with that of Frazier (8.3 per cent). It is indicated rather in those cases in which clinical signs suggest the existence of an expansive process of the angle.[5]

Section of the descending fibers of the trigeminal tract in the medulla oblongata should be the ideal method because the sense of pain and temperature are abolished, without loss of the sense of touch; subjective numbness is practically absent; in all instances the motor root is preserved; facial weakness and keratitis do not occur.

Although most of the disturbances, particularly those of gait and station, and paralysis of the vocal cords, which occurred in a considerable portion of the earlier cases in which the incision was made at a level corresponding to the border between the middle and the inferior third of the olivary eminence, could be avoided by sectioning the tract at the level of the obex, or caudal to it, this operative method still has undesirable effects that limit the field of application. These are principally: a higher operative mortality compared with the Frazier method; longer hospitalization; relative frequence of recurrence.

Bearing in mind that trigeminal neuralgia usually affects older subjects, often those with arteriosclerosis and hypertension, it will be understood that this surgical measure cannot be used systematically for all patients. It should be reserved for those who are young or middle-aged, and in good general condition, for whom preservation of the tactile sensation in the face is desirable, and in whom, in case of recurrence, a secondary operation some years later could be performed without danger to life.

For patients suffering from particularly severe neuralgia, in whom subsequent recurrences are most likely to occur, tractotomy is not recommended, nor in those cases in which the neuralgic pain is exclusively, or principally localized in the 3rd trigeminal division. This is not because it is difficult to obtain analgesia in this territory, but because it is potentially more dangerous.

In conclusion, tractotomy is indicated in those patients who are young, and in good general condition, with moderately severe trigeminal neuralgia especially if localized in the ophthalmic and maxillary areas. (Indications for tractotomy in cases of bilateral trigeminal neuralgia will be discussed in a later paper.)

SUMMARY

A total of 124 patients with major trigeminal neuralgia have been subjected to section of the descending fibers of the trigeminal tract in the medulla oblongata. Because of a modification of the original operation, the patients fall into two groups: those (40) in whom the trigeminal tract was sectioned at a level corresponding to the border between the middle and the inferior third of the olivary eminence; and those (84) in whom it was per-

formed at a level corresponding to the lower end of the 4th ventricle. This modification has practically eliminated the danger of laryngeal palsy, and the disturbances of gait and station.

Considering that the trigeminal tractotomy is an intervention which is potentially more dangerous than section of the sensory fibers by the subtemporal approach, and because it is followed by a relatively great number of recurrences, it cannot be used as a routine measure. It is indicated in a restricted number of subjects in whom preservation of the tactile sensation in the face is desirable, and in whom, in case of recurrence, a secondary operation can be performed without risk to life.

Tractotomy is thus indicated in young and middle-aged subjects, in good general condition, and with moderate neuralgia, especially if localized in the ophthalmic and maxillary divisions.

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ROENTGEN THERAPY SUPPLEMENTING SURGERY IN THE TREATMENT OF GLIOMAS

G. M. TICE, M.D., AND N. W. IRVING, M.D.
University of Kansas School of Medicine, Kansas City, Kansas

(Received for publication March 6, 1950)

IN CONSIDERING the effect of radiation on brain tumors several problems present themselves. First we must know that we are dealing with a brain tumor. No series dealing with brain tumors is of any statistical value unless there is microscopic proof of the presence of a neoplasm. In reviewing the literature we found several series of cases reported in which this evidence is lacking. It is generally known, for instance, that certain inflammatory processes may produce a picture simulating an obstructing brain tumor. Since we recognized the fact that surgery must be done in order to make a positive diagnosis, we are compelled to take into consideration the effect of the surgical procedure on any improvement that the patient may show. This improvement may be due to the removal of a large part of the tumor or it may be due only to the decompression incidental to the surgical procedure. As far as curing the tumor is concerned, we are of the opinion that the only significant radiation dose recorded is the tumor dose. Several series of reported cases were found in the literature, particularly prior to 1935, that did not accurately indicate the tumor dose administered. The question then presents itself as to the proper dose necessary to destroy a deep-seated brain tumor, or in the case of a glioma where destruction perhaps is not possible by means of radiation, we are interested in the dose that will prolong life appreciably and add to the comfort of the patient during this period. Finally we are faced with the problem that presents itself whenever radiation is used on a deep-seated malignant tumor; this is the question of the tolerance of normal tissue to radiation. It must be decided, then, in the interest of the patient whether this tolerance dose will afford him relief from the presenting symptoms or will produce other symptoms that will make him more uncomfortable. It seems to the authors that in no other location in the body is it more advisable to gauge the dose with accuracy than when administering radiation for a brain neoplasm.

It is not our purpose in this presentation to contribute any new ideas on the method of therapy, or to present data on animal experimentation. We have, over a period of 15 years, been referred the usual variety of brain tumors seen in a moderately active Neurosurgical Service in a general hospital Medical School. The survey was made largely as a means of evaluating for ourselves the results obtained by our efforts in radiating brain tumors. Naturally our interest was drawn at the same time to the results obtained by others and the methods used by others as compared to our own procedure. Certainly as we look over our results in terms of length of survival, we must be impressed with the fact that in most cases where the brain

tumor could not be removed our hope in giving roentgen therapy was in the interest of palliation.

It is difficult to evaluate the effect of radiation on brain tumors. The size of the tumor can usually not be determined by direct visualization, if it is an inoperable tumor. Whether it is decreasing or increasing in size cannot be determined except by indirect means. In all of the cases presented the patients have had surgery prior to radiation. The radiologist was called upon because the surgeon felt in the case of the small tumor that some malignant tissue may have been left behind. In the case of the massive infiltrating tumor, he had removed perhaps only a small bit for biopsy. Undoubtedly an immediate good result in the general well-being of the patient followed the decompression if nothing else was done. This makes it, of course, difficult to evaluate benefit received by the patient who subsequently received roentgen therapy following this decompression procedure. We can best demonstrate the effect of radiation in the case of recurrent tumor. A patient has had, perhaps, a series of x-ray therapy following decompression then, after a period of a few months, there is definite symptomatic evidence of recurrence. In this individual surgery is not done on the second admission and any benefit received can be attributed to the radiation. There is also the case of the medulloblastoma in which the sutures are widespread, the skull is large, and there is obvious evidence clinically of increased pressure. Following radiation this rapidly growing tumor also regresses rapidly and the size of the skull actually becomes less in circumference, indicating again the direct effect of radiation on this particular type of tumor.

Our method of treatment has varied a great deal over the years as our knowledge of the tolerance of x-ray by normal tissue has been gained, as we have learned the dose necessary to destroy malignant tissue, and as we have procured equipment designed to deliver progressively shorter wave lengths. Most of the patients reported have been treated with 200 KVP and a wave length of H.V.L. 0.9 mm. copper. For the last 2 years we have had 250 KVP at our disposal and we now use routinely filtration to produce a wave length of H.V.L. 1.7 mm. copper. At present the average brain tumor in the adult receives a dose of 5000 r in 1 series or the treatment may be, if indicated, increased to 6000 r in 2 series spaced 2 months apart. This dose in the child is reduced to 4000 r. The total dose is given in 25 or 30 treatments. There is a treatment daily, except Sunday. One portal daily is used. Usually 4 portals are utilized to cross-fire the tumor area. If the tumor is located in the occipital region the portals consist of each lateral, posterior, and vertical. Sometimes it is feasible to divide the posterior portal into a suboccipital and a vertical occipital portal. It hardly seems feasible in a large skull to treat an occipital lobe tumor through a frontal portal. The size of the portal will depend largely on the size of the skull and the size of the tumor. Our usual portal measures 8 by 8 cm. or 10 by 10 cm. The daily dose of 300 r measured in air is used. We have not hesitated after a series as described above to repeat the treatment in from 4 to 6 months if the patient is seen to be deteriorating.

A number of articles can be found in the literature prior to 1935 in which an attempt has been made to evaluate radiosensitivity of brain tumors. It is the authors' opinion that there has been such a change in the quality of ray used since 1935 that it is hardly feasible to compare results before that date with those obtained now. A brief partial review of the subject starting with the literature in 1929 will summarize the opinion of various authorities as to the value of radiation therapy in the treatment of gliomas. Ewing[9] in 1921 stated that undifferentiated tumors should be sensitive to radiation. If this statement is accurate then gliomas, he thought, should be somewhat radiosensitive and such tumors as neurosarcomas and neurofibromas should be radioresistant. In 1925 Bailey[2] concluded that gliomas react very irregularly to radiation. Pancoast and Grant[19] in 1926 analyzed 125 cerebellar tumors and decided that surgical procedure rather than radiation accounted for any clinical improvement seen. Pancoast[17] in 1927, after studying 90 cases of brain tumor, decided that no clinical improvement could be seen from radiation except in 1 or 2 cases. Hyslop and Lenz[12] after studying a group of 19 cases of glioma were enthusiastic about the results obtained from radiation. An attempt was made in 1928 to correlate the fact that radiation had been given with the subsequent microscopic picture. This study was done by Bailey, Sosman, and Van Dessel.[4] They concluded that no improvement was noted that could be detected on their microscopic sections. In 1928 Pancoast[18] stressed the fact that since normal brain tissue is radioresistant larger doses of x-ray can be used without fear of damaging the normal tissue. It was his conclusion, after study of 48 cases of brain tumor, that there had been some improvement by radiation supplementing surgery. Sargent and Cade[24] in 1931 felt that radium could be used to some advantage in the treatment of brain tumors. Deery[7] in 1936 attempted to correlate the clinical and histopathological changes following radiation in a group of 50 cases. Only cases were chosen in which pre- and postoperative specimens were available. All types of gliomas were included but medulloblastomas, glioblastomas, and astrocytomas predominated. Histopathological change was seen in all types studied, both those of low differentiation such as medulloblastomas and those well differentiated such as astrocytomas. The outstanding changes noted consisted of increase in necrosis, reduction in cellularity, reduction in mitotic figures and appearance in some cases of giant cell forms.

A moderate amount of experimental work has been done by a few authorities on animals in an attempt to determine the amount of radiation that the brain can tolerate and the amount of radiation necessary to destroy a tumor in the brain. Davidoff, Dyke, Elsberg, and Tarlov[6] reviewed work done prior to 1938 on radiation of the normal brain and described their work on rhesus monkeys. In their experiments a skull flap was removed and x-ray was applied directly to the brain. The dose was given in a relatively short period of time. It was found that a dose of 4000 r or more to a cerebral hemisphere was followed by a more or less well marked paralysis of the contralateral limbs. A few months after a dose of 2000 r to 3000 r hemiparesis

developed in some of the animals. In 2 animals that received 4000 r and 5000 r respectively to the cerebellum, disturbances developed after from 3 to 5 months. Histologically the effect of massive doses of roentgen rays was especially marked in the glia and nerve tissues. Changes in blood vessels were slight.

C. M. Wilson of the Physics Department of Westminster Hospital Medical School[28] studied the changes taking place in the brains of animals after radiation. He radiated the left side of the skull of 8 rabbits with a single dose of 2850 r. About 100 days later neurological changes began to occur. One of the animals became blind. At autopsy the cortex immediately adjacent to the heaviest radiation showed no change, but deep in the brain there were foci of necrosis without vascular change. The left lobe of the cerebellum, even though not radiated, showed marked changes in both the cortex and white matter. All animals when killed showed similar focal necrosis with particular damage seen in the cerebellum.

A unique scientific experimental approach to the subject of the value of roentgen therapy to brain tumors was made by Tansley and Wilson.[26] They were able to produce experimentally sarcoma and gliomata in mice by the direct installation of a small quantity of methylcholanthrene powder into the brain. The gliomata so induced were transplanted in other mice. These transplanted tumors were then irradiated. They were not able to produce any cures but definite regression was seen in some of the tumors. In general the higher doses were more effective in arresting tumor growth. A maximum dose of 3247 r was used.

Attempts have been made by various authors to evaluate the effect of radiation on normal and neoplastic brain tissue as seen clinically or in the pathological laboratory. Alpers and Pancoast[1] in 1933 studied 22 cases of gliomas that had received radiation therapy. In medulloblastoma they observed a patchy type of tumor cell destruction. They saw healthy and dying tumor cells side by side. They found no effect on the more differentiated tumors. They summarized the effect of irradiation on normal brain tissue as follows: There is fatty degeneration of the cells of the cortex and in the Purkinje cells of the cerebellum. Vessel thickening was not observed. It is interesting to note that because of their observation of minimal normal brain damage they were impelled to say in 1933, "A full erythema dose over three portals with saturation, say up to 200% would seem to be the very best that we can ever hope to apply by external radiation."

In 1937 Frazier, Alpers, Pendergrass, and Chamberlin[10] made a study of 30 cases of irradiated gliomas. They divided the cases for purpose of study into groups receiving less than 1000 r, calling this an inadequately treated group; those receiving 1000 r to 2000 r which they called the moderately treated group; and those receiving more than 2000 r which they described as their adequately treated group. The assumption must be made that they considered 2000 r sufficient. One of their patients received a little over 7000 r in 5 series, but most of them were treated with a dose of from

2000 r to 3500 r in this group. In the same article they make the statement that, "Irradiation has not been able to cure any of the many types of glioma, but it has exerted some influence on the tumors mentioned, particularly on the medulloblastomas." If it is true, and the fact is generally accepted now 12 years later with more refined radiation technic, that we cannot cure a glioma with radiation, then we must modify our thinking of an adequate dose to that dose which will be just short of that which will damage the normal brain structure enough to produce symptoms and yet will produce the greatest damage to the tumor. It was their conclusion that the larger doses were necessary. In their series response was seen in the medulloblastoma group. Slight response was seen in the astrocytomas. Very little response was noted in glioblastoma multiforme and none in the oligodendroglioma group. Their opinion of improvement was based on histological study before and after treatment.

O'Connell and Brunschwig in 1937[16] used a scientific approach to the subject by selecting a normal brain, a brain containing a tumor that had not been irradiated, and 3 brains that had received irradiation. The doses used in the 3 cases were 12,645 r, 15,500 r, and 14,229 r. These figures represented total air dose. Tumor dose was not stated. No changes in any way comparable to those found in the irradiated brains were demonstrated in the 2 control brains. In the cerebral cortex and to a lesser extent in the cerebellum structural alterations were numerous. The nerve cells showed retrogressive changes. In many the cell body was swollen and rounded with nucleus displaced toward the periphery. There was an excess of lipochrome pigment. Some nerve cells were shrunken with darkly staining nuclei. No change in nerve fibers was noted. Interstitial tissue in the cortex showed marked degenerative changes. Perivascular lymphocytic infiltration, arterial hypertrophy, hyalinization of the media and swelling of the intima were noted.

Wachowski and Chenault[27] treated 3 patients with doses varying from 7750 r to 8910 r. In none of their cases was the tumor entirely destroyed. They report following the above doses that, "Pathological changes are wide spread and affect all cellular elements, nerve cells degenerate and excessive amounts of fat accumulate. While degenerative changes are seen about smaller blood vessels, the larger vessels were undamaged." They felt that the vascular effect of radiation was not the predisposing factor in deterioration of nerve tissue.

As a result of the observations of Bailey, Sosman, and Van Dessel,[4] of Sachs, Rubinstein, and Arneson,[23] of Kaplan,[14] of O'Connell and Brunschwig,[16] of Sosman[25] and others, it has been generally accepted that if any palliation is to be expected without excessive normal brain damage, the dose administered to the tumor should be, in the child from 3000 r to 4500 r, and in the adult when given in 1 series up to 6000 r. Kaplan would, in the adult, deliver in most instances 6000 r and he feels justified in risking a dose of 9000 r given in 1 series in a period of 1 month. It was his opinion

that in a series of 10 patients so treated there had been no clinical evidence of brain damage. In this series he did not, however, get a tumor dose above 6000 r. When Dyke and Davidoff[8] made inquiry about therapeutic radiation, prior to publication of their book in 1942, they found that Pendergrass was delivering up to 6500 r to a midline glioma and Brunschwig and Hamann were giving 5000 r to medulloblastoma. Many cases are recorded of extremely large doses of x-ray administered over a period of time. In most of the cases the depth dose used is not adequately recorded. Fried[11] reported 2 cases of glioblastoma multiforme that received total radiation of 32,649 r and 22,800 r respectively. He does not state the depth dose. In Fried's cases the patients were both alive at the time of the report. The one receiving the largest dose was able to ride a bicycle and do some work. He had, at the time of the report, been alive for 2 years and 10 months. The other had gone 3 years and was in fair condition. Kaplan[14] has reported the highest recorded depth dose that we were able to find. He gave a total dose of 25,200 r to the tumor, a glioma, over a period of 2 years. The patient lived 4 years.

Jenkinson and Oldberg[13] classify those who treat brain tumors into two groups. One group is extremely radical, removing large segments of the brain with no regard to ultimate function. Their only consideration is the removal of the tumor. The other group is less radical in the surgical approach, recognizing the fact that life may not be worth living, even though it is prolonged a bit, if the surgery is too radical. This is the group to which Jenkinson and Oldberg subscribe and we certainly go along with them in their approach to the problem. They believe "that many times it is better to return a person to society after a short and comparatively inexpensive hospitalization, even those whose recurrence interval may be short, than to indulge in an operative tour de force and produce an aphasic or hemiplegic individual whose survival period at best is but little longer." They treat a temporal lobe tumor with a dose of 4500 r measured in air delivered through 5 portals. The depth dose is not stated. The series is repeated in 2 months. If there is a recurrence it is again repeated in 6 months. They do not give survival figures but state that "A patient's life can usually be prolonged for some years in comparative comfort and with control of his faculties by the use of conservative surgery followed by irradiation."

It is difficult to evaluate the effect of irradiation following surgery on brain tumors. Repeatedly we find the statement made in literature that 5-year survival rates mean but little. This may be true but we know of no better way to indicate our results than in terms of survival over the years or to indicate the number of months that the patient with a certain type of tumor lived whose survival was short. We have followed the lead of McWhirter[15] in listing our few cases of brain tumors in certain groups. In other cases we have varied from his classification.

The astrocytoma is usually a well circumscribed tumor that lends itself to surgical removal. In our series of patients treated by radiation postoperatively 30 per cent are alive 5 years later. It is difficult to know how

much, if any, of this survival percentage is due to the irradiation. We have assumed that the tumor in each case was not completely removed but one has to wonder, for instance, in our case that has been followed for over 12 years, and the patient appears to be completely cured, if this survival time is not entirely due to the surgical procedure. Our record shows that this particular patient received the least amount of radiation of any of our surviving patients. While astrocytoma is not considered by most authorities to be a radiosensitive tumor, histological evidence has been obtained to show that in some cases there is definite effect from radiation. We see no reason to refuse treatment in any case in which removal is reported as incomplete. Conversely one might state that if the surgeon is sure the tumor has been completely removed there is no reason for subjecting the patient to radiation therapy.

Cellular meningiomas and hemangioblastomas are classed together following the lead of McWhirter.[15] He considers them radiosensitive tumors and differentiates them from the medulloblastoma group by calling them "non-metastasizing." In this group of 5 tumors we have 20 per cent 5-year survival. The series is too small to justify any conclusions. Of the 2 patients living, 1 is a young woman in whom an incomplete removal of a left frontal lobe tumor was done. She received 2 series of irradiation with a total depth dose of 6400 r. She is free from symptoms 6½ years after therapy.

Glioblastoma, spongioblastoma and oligodendroglioma are classed together in one group of relatively insensitive brain tumors. None of our patients has survived 5 years, although 3 have been followed for 3 years and are in fair health. Unfortunately these tumors are infiltrating and defy, usually, complete surgical removal. One is always tempted in the hopeless case of this type to forget all warnings about damage to the normal brain by radiation and extend the depth dose above that usually considered safe. It is only by heroic radiation therapy that we can expect to improve our results in this group and we must face the fact that even this effort will very likely be of no avail.

Medulloblastoma and malignant choroidal papilloma might be included in a group of radiosensitive metastasizing tumors,[15] but it is only in this respect that they are similar. For purposes of comparison with other figures we have considered the medulloblastoma by itself. We had but 1 case of choroidal papilloma. Medulloblastoma is primarily seen in childhood, but may occur in the adult. Its common site of origin is in the 4th ventricle and cerebellum. The problem of control differs from that of other brain tumors because of its tendency to disseminate cells throughout the subarachnoid spaces of the spinal canal and brain. It is generally recognized as a radiosensitive tumor, being likened to the lymphoblastoma in this respect by some authorities. In all of our cases a decompression was first done and as much of the tumor as possible was removed. This was followed by irradiation. Peirce, Cone, Bouchard and Lewis[20] advocate aspiration biopsy of suspected medulloblastoma followed by irradiation as the treatment of

choice. Cutler, Sosman and Vaughan[5] favor radiation of suspected cerebellar tumors without biopsy because of operative risk. It seems to the authors that the possibility of a mistaken diagnosis without biopsy and the necessity of decompression make it imperative for surgery to be done first in this group of cases. Our average survival time in the medulloblastoma group is 21.3 months. We have 3 patients living 3 years; 1 of these is a man who was 31 years old when the diagnosis was made. An average dose of 7500 r has been used in these 3 cases. It is our routine to deliver from 1500 r to 2000 r air dose to the entire spine with a 10 by 20 cm. portal in tumors of this type.

TABLE 1

Summary of entire series

Pathology of Tumor	Total Cases	Total Living	Total Dead	Average Survival of Dead (mos.)	Average Survival for Entire Series (mos.)	Percentage of Entire Group Living Over Stated Number of Years				
						1 yr.	2 yrs.	3 yrs.	4 yrs.	5 yrs.
Astroblastoma Astrocytoma	13	5	8	28	46.4	77%	61%	61%	39%	39%
Cellular Meningioma Hemangioblastoma	5	2	3	6	24	40%	40%	20%	20%	20%
Glioblastoma Spongioblastoma Oligodendroglioma	23	4	19	12	16	51%	21%	17%	8%	0%
Medulloblastoma	10	3	7	16	21.3	60%	50%	20%	0%	0%
Choroidal Papilloma (malignant)	1	1	0		61					100%
Total	52	15	37		28.6					

Survival rates quoted by other physicians are as follows: Bailey and Cushing[3] reported without radiation following surgery an average of 7 months and with radiation the average was increased to 19 months. Dyke in 1942[8] reported 22.3 months survival average. Peirce, Cone, Elvidge, and Tye in 1945[21] reported 22 months. McWhirter[15] in his group reported a 14 per cent 5-year survival.

In our 1 case of malignant choroidal papilloma the patient has survived 61 months and shows no evidence of recurrence of her tumor. Another case, of hemangioma proven by operation in 1929, perhaps should not be included in this series but we have observed amelioration of his symptoms following radiation therapy on 2 occasions.

Referring to Table 1 it will be seen that of our total of 52 patients seen from June 1933 to January 1, 1949, 15 are living and 37 are dead. The highest percentage of survival is in the astroblastoma and astrocytoma group. The lowest is in the group that includes the glioblastoma, spongioblastoma, and oligodendroglioma.

Table 2 shows the length of survival in months of each individual now living and the amount of radiation given. In these cases an average of 5900 r was delivered to the tumor. In the cases in which the patients are not now living not much can be gained by summarizing the tumor dose. Some of these patients were treated prior to 1935 and had recorded tumor doses of as low as 2000 r or 3000 r. In the group that is now dead we find the case of an astrocytoma in a 29-year-old man who received a total of 12,000 r tumor

TABLE 2

Summary of Living cases

Name of Patient	Age in Years	Pathological Diagnosis	No. of Series	Total r to Tumor	Location	Type of Tumor Removal	Number of Months Living Since Diagnosis
B.B.	7	Astroblastoma	3	7500	Lt. cerebrum	Partial	63
L.B.	6	Cystic Astrocytoma	2	4800	R. cerebellum	Partial	96
A.McK.	10	Astrocytoma	1	1200	Lt. cerebrum	Complete	150
R.B.	45	Astrocytoma	3	7200	R. temporal	Partial	46
R.B.	7	Astroblastoma	2	5200	R. temporal	Partial	43
I.W.	50	Hemangioblastoma	1	4500	Lt. frontal	Partial	24
B.M.	28	Cellular Meningioma	2	6400	Lt. frontal	Partial	78
R.S.	8	Medulloblastoma	3	7200	Post. fossa	Partial	30
J.Z.	7	Medulloblastoma	2	7200	Lt. post. fossa	Partial	37
L.L.	31	Medulloblastoma	3	8100	Post. fossa	Partial	36
B.P.	14	Glioblastoma	2	8000	Lt. occipital	Partial	42
H.G.	53	Glioblastoma	1	4200	Lt. temporal	Partial	8
R.M.	3	Glioblastoma multiforme	3	4600	4th ventricle	Partial	46
S.S.	14	Spongioblastoma	2	5400	4th ventricle	Partial	20
G.W.	36	Hemangioma (proven hemangioma by surgery 1929)	3	9000	R. cerebrum	Partial	20 yrs.
B.T.	11	Malignant Choroid Papilloma	2	7200	Choroid plexus	Partial	61

dose in 6 series for a left frontal lobe astrocytoma. He died after a 79-month survival.

A paper of this type must make mention rather briefly of that series of cases in which no biopsy was obtained, but in which the surgeon felt sufficiently sure of the diagnosis of brain tumor from history, physical findings, and ventriculograms, that the patient was referred for x-ray therapy. We have 23 cases in this group. Ten of these patients are still alive, 13 are dead. The histories of 2 cases in this group are related as a matter of interest.

Case 1. F. C., male, aged 17, was admitted to the medical Service of the University of Kansas Hospitals on May 29, 1941. In February, 1941 he first noticed that his right eye became tired when he read a great deal. He was examined by an ophthalmologist who discovered a papilledema. Vision was normal. About 4 weeks later he had a very severe headache which lasted all day. After that he had two or

three slight headaches which came on when he used his eyes excessively. He had vomited a few times. His past history recorded scarlet fever and a tonsillectomy was done at the age of 7.

Examination. Pupils reacted equally. Fundi showed bilateral papilledema of 3 D. There was a slight difference in activity of reflexes on the two sides, with hyperactivity on the right. Laboratory findings were within normal limits.

Ventriculograms revealed marked dilatation of the lateral ventricles and 3rd ventricle. It was felt that there was a midline obstruction in the aqueduct.

Operation. On June 9, 1949 posterior fossa exploration was done. A block was found in the aqueduct from the 3rd to the 4th ventricle. The nature of the block was not revealed. Exploration was not deemed advisable.

Course. Following operation x-ray therapy was instituted. A dose of 5,000 r to the midline was delivered. This patient rapidly became free of his symptoms. At present he is a student in a medical school.

We think this is undoubtedly a case of aqueduct stenosis; in fact, stenosis was found in operation and there was a question of the possibility of a tumor surrounding the aqueduct. It is doubtful if radiation had anything to do with this boy's rapid recovery. Undoubtedly the relief of the stenosis at operation was the factor bringing about a cure.

Case 2. M. P., male, aged 15, was admitted to the Neurosurgery Service on Nov. 24, 1943. In February severe headaches developed with numbness on the left side of his face. His mouth drew to the left side. In May 1943 he started having numbness of the feet and stumbling gait. He lost almost all vision and there was ptosis of the left lid. He was told by his family physician that he had a brain tumor. A few x-ray treatments were given which relieved his headaches for a while. For the last 6 weeks he had not been able to stand.

Examination. There was diplopia. The left temporal field was contracted. Optic discs showed pallor but no papilledema. The palate and tongue deviated to the left. The legs showed muscular atrophy below the knee. Knee kicks were absent. Babinski was negative. There was absence of pain and temperature sense below the middle third of the leg. There was also absence of deep touch and vibration in this same area.

Course. A diagnosis of medulloblastoma was made from the clinical examination without biopsy. X-ray therapy was instituted.

The patient was readmitted on April 16, 1944. At this time he was able to move his legs but could not yet walk. Extraocular movements were normal and discs were clear. The tongue still deviated slightly to the left. Atrophy of muscles of the legs with absent reflexes was noted. Again x-ray therapy was instituted. In January 1946 the patient returned. He had no headaches. He still had diplopia on looking to the right and up. There was no vomiting. Upon recent examination he was able to walk with braces. Other symptoms were gone.

This case has been carried in our files as a case of medulloblastoma. The patient has improved apparently under x-ray therapy. We have to doubt the diagnosis since there has been no microscopic proof and since the patient has lived 5 years. He received a total dose of 7200 r to the brain midline.

SUMMARY AND CONCLUSION

Fifty-two patients with proven brain tumors have been treated with x-ray therapy during the 16 years prior to January 1, 1949. Of this number 15 are living and 37 are dead. The astroblastoma group shows an average survival rate of 46.4 months with 30 per cent alive for 5 years. The cellular meningioma and hemangioblastoma group is too small to justify conclusion; 1 out of 5 is alive for 5 years. The glioblastoma, spongioblastoma and oligodendroglioma show no 5-year cures; there is an average survival of 16 months. The medulloblastoma series, consisting of 16 cases, shows a 21.3 months average survival, and no patient living for 5 years.

Evidence has been submitted that radiation of the brain of animals and of men may produce degenerative changes in the normal brain.

Since we likely cannot cure glioma with radiation our objective must be such a dose that the tumor will show maximum regression with the least amount of damage to normal brain tissue.

There is a justified tendency toward a higher dose. Our living patients received an average dose of 5900 r to the tumor. It is our impression, gathered from the literature, that from 5000 r to 6000 r is the generally accepted tumor dose protracted over from 20 to 30 treatments.

All cases reported above were referred to us by the Neurosurgical Service of the University of Kansas Medical Center by Dr. Frank Teachenor and associates.

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GLIOMAS OF THE NEUROHYPOPHYSIS AND HYPOPHYSIAL STALK

A PRELIMINARY REPORT

NILS ANTONI, M.D.

Neurologic Clinic, Serafimerlasarettet, Stockholm, Sweden

(Received for publication March 6, 1950)

THE correlation between acromegaly and eosinophilic hypophysial adenomas was already old news (Benda 1901) when Cushing discovered the correlation between the equally peculiar syndrome named after him and the basophilic cells of the hypophysis. Vigilant and unbiased observation has shown that the correlation between the first two is not entirely absolute, and even less absolute between the second two. Nevertheless both syndromes are still regarded as the manifestations of specific endocrine hyperfunction. It has been known since the report of Babinski in 1900 that adiposogenital dystrophy may be the result of a hypophysial tumor. This combination more often referred to as Fröhlich's syndrome, is generally considered to be the result of diminished activity of the hypophysis, i.e., hypopituitarism. It has been a widespread opinion that one of the main causes of hypopituitary syndromes are chromophobic hypophysial adenomas, i.e., tumors formed of the chromophobic or chief cells of the pituitary gland proper. This doctrine was first advanced by Norman Dott and Percival Bailey,[4] in a lecture in 1924. These authors made a broad attack on the whole question of hypophysial adenomas, analyzing all the previous research and the extensive data from 162 cases of tumor, 107 chromophobic, from the Cushing clinic. During the quarter of a century since their report, the theory of the chromophobic adenomas and their particular topographic, histologic and clinical characteristics has been blindly accepted and few attempts have been made to check its veracity. The few control studies (Kraus,[8] Roussy and Oberling[16]) have been mainly confirmatory in nature. The general conception laid out by Dott and Bailey was so amazingly simple and schematic that it is not surprising that it won such immediate and general recognition.

Their theory was roughly as follows. There are three kinds of hypophysial tumors, all deriving from the pars anterior, the pituitary gland proper. One kind, which is rare, comes from the smallest cellular component of the adenohypophysis, the basophilic cells; these basophilic growths are always small, seldom grow beyond the sella and therefore cause no symptoms due to pressure on adjoining structures. Much more common are the eosinophilic growths, originating from the eosinophilic cells occurring in large numbers in the normal adenohypophysis. These not seldom grow up out of the sella, become suprasellar in other words, and exert pressure on neighbouring structures, particularly the optic chiasm. The most common of all, accounting for 80 to 90 per cent of the hypophysial tumors, are the

chromophobic adenomas coming from the largest cellular component of the anterior lobe, the chromophobic or chief cells. It is these tumors that far the most often break through the sellar diaphragm and spread out in the suprasellar space, resulting in pressure on the chiasm, optic nerves and tractus opticus and infundibulum, tuber cinereum and other structures.

This theory does not explain the numerous and practically important topographic and clinical peculiarities of the tumors which the Cushing school call chromophobic adenomas. Nor does it leave any room for tumors

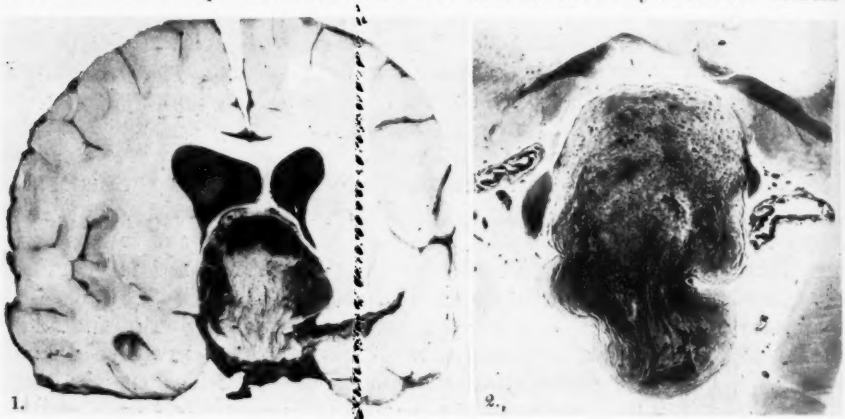

FIG. 1. *Case 1.* Tumor projecting up between the two temporal lobes, pushing them apart and filling the 3rd cerebral ventricle. Intrasellar portion removed. Many blood and colloid cysts.

FIG. 2. *Case 7.* Low-power view (×1.6). A lace-groove is visible, corresponding to the limit between the intra- and suprasellar portion of the tumor. The tractus optici are visible on both sides of the suprasellar portion.

arising in the neurohypophysis. "Primary tumors of the posterior lobe are practically unknown," said McLean.[11]

I have now made a study of the tumorous tissue in 10 cases diagnosed as chromophobic hypophysial adenoma at the Neurologic Clinic in Stockholm. In 2 cases the study was made post mortem with the tumor and brain *in situ* (Figs. 1 and 2) and in the other 8 the tissue removed at operation was examined. This study has shown that the opinion that chromophobic hypophysial adenomas constitute the bulk of the suprasellar tumors connected with the hypophysis needs revision, to say the least. Not less than 8 of the 10 tumors were undisputably or almost certainly *gliomas of the ependymal type* (Figs. 1–8, 12), corresponding most closely to the ependymomas of the Cushing-Bailey classification.

These tumors were highly cellular. The cells were well individualized as a rule, mostly elongated and arranged in sinuous garland-shaped strands in some places and in other places in rounded groups or acini sometimes separated by thin, often vascular connective-tissue membranes. These ependy-

mal-like cells often tapered off at one end and supported themselves with their narrow ends against capillary or precapillary blood vessels, forming corona-like figures around them. Many places showed rosettes or pseudorosettes grouped around narrow cavities. Cilia were not uncommon. I was not able to discover undisputable blepharoplasts. Nor did I see any multinuclear cells or mitosis. There was no necrosis but most of the tumors contained cysts with "colloid" contents. The cysts developed in the same way as in certain

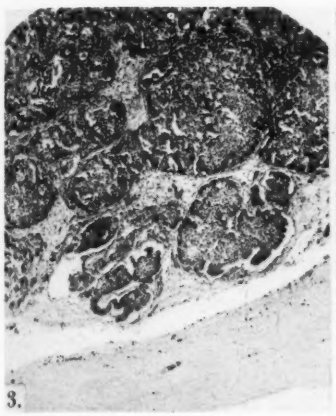

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FIG. 3. *Case 1.* Pseudo-alveolar structure. Heidenhain's iron-alum-hematoxylin stain (×30).

FIG. 4. *Case 1.* Heidenhain's iron-alum-hematoxylin stain (×83). "Many scattered vascular channels, surrounded by clear areas" (McLean, ependymoma, *Bumke u. Foersters Handb. Neurol.*, 1936, *14*: 154).

gliomas and neurinomas, viz., through "hyaline degeneration" of the matrix or exoplasm and confluence of the degenerated products to form single or multiple cysts of various sizes. (Figs. 7 and 8).

The 2 autopsied cases (Figs. 1 and 2) are particularly instructive. In both instances the tumor was composed of a small intrasellar portion and a much larger suprasellar portion, the latter projecting up between the two temporal lobes, pushing them apart and replacing the 3rd cerebral ventricle. The suprasellar portion lay subarachnoidally and was coated outermost with a thin connective-tissue membrane, a pia mater. Under this membrane was a thin layer of tissue built up in the same manner as the central nervous system, with mostly glia cells and a maze of glia fibrils. In some places this layer sent regular septa a short way into the tumor mass. There is no doubt that the suprasellar portion of the tumor was composed of a strong, neoplastically thickened hypophysial stalk. There was no capsule on the intracerebral crown of the tumor, the neoplastic tissue there merging without a sharp line of demarcation with the surrounding brain tissue.

Six of the operatively removed specimens were undisputable or almost

undisputable ependymomas. One of the other 2 contained a highly cellular tissue which was perhaps of gliomatous nature. In the other case, it is impossible for me to identify the nature of the tissue; it was of loose structure, as if vacuolated, and I have no idea what it was.

What is particularly noteworthy is that in most of the cases in which the neoplasm was identified as an ependymoma, the clinical picture corresponded in every respect with the "hypopituitarism" considered to be distinctive of the so-called chromophobic adenomas (Fig. 11).

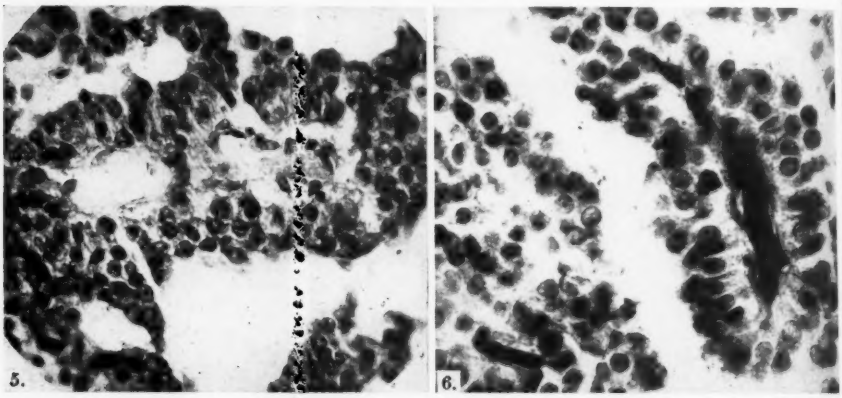


FIG. 5. *Case 4.* Ependyma-like, folded bands of closely packed cells. Vascular channels, pseudorosettes and cilia are visible. Iron-hematoxylin stain (×370).

FIG. 6. *Case 7.* Papilla formation with central vessels. Heidenhain's iron-hematoxylin stain (×515).

Let us assume for the moment that the majority of the suprasellarly growing non-eosinophilic tumors connected with the hypophysis or its stalk are really gliomas, mostly ependymomas, arising from the neurohypophysis or hypophysial stalk. We then have the explanation for a large number of the puzzling topographic and clinical features previously mentioned.

1) This assumption would explain why they grow up out of the sella turcica and spread out in the suprasellar region much more often than other hypophysial tumors. They have a preformed route of propagation, the hypophysial stalk. It may also be that the tumor sometimes starts in the stalk, grows first suprasellarly and afterward down into the sella turcica. For that matter, it could happen that the tumor never extends down into the sella, but remains entirely suprasellar. (This should also be possible in the case of real adenomas originating in the anterior lobe, for the pars tuberalis of the latter coats the hypophysial stalk.)

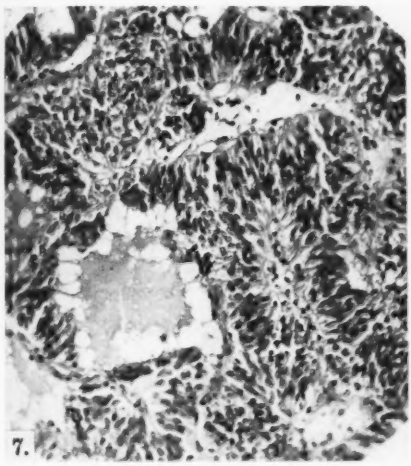
2) It would explain why they much more often than eosinophilic adenomas cause pressure on the optic chiasm and the region of the tuber cinereum, with resultant defects in the visual fields and other disorders.

3) It would explain why they much more often than the eosinophilic adenomas are accompanied by little or no roentgenologic enlargement of

the sella turcica or other roentgenologic changes in this space. For one thing, it would explain why the "niche" projecting from the anterior part of the sella so common in the roentgen picture accompanying eosinophilic adenoma is lacking in these cases. If these tumors started in the pituitary gland proper, there is no reason why they, too, should not dig themselves a niche.

4) It would explain why the endocrine vegetative disorders accompanying these tumors are entirely, or at least mainly, privative, the result of hypopituitarism.

5) It would explain why this endocrine vegetative syndrome does not occur in every case. The latter circumstance was mentioned by Dott and Bailey[4] and has been confirmed occasionally in the Neurologic Clinic. The obvious explanation for this irregularity is that the tumors vary as regards



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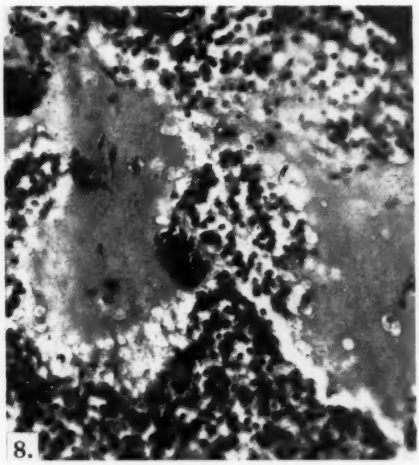

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FIG. 7. *Case 6.* Abundant cyst formation. Hematoxylin-eosin stain (×110).

FIG. 8. *Case 7.* Cyst formation through "hyaline" or "colloidal" degeneration of the tissue. Heidenhain's iron-hematoxylin stain (×200).

the direction in which they grow, the region they occupy and the size they acquire.

6) It would explain why these tumors are much less responsive to roentgen treatment than the eosinophilic variety. Mature gliomas are generally insensible to the roentgen ray.

7) It would explain the missing tumors from the neurohypophysis and hypophysial stalk. Here they are!

Ependymomas occur in different frequencies in different parts of the central nervous system. They account for *only 2.7 per cent of the Cushing and Bailey* series of cerebral gliomas but for as much as 46 per cent of the Kernohan *et al.*[7] series of intramedullary spinal tumors. I have seen them several times in the cauda equina, where they probably start in the filum

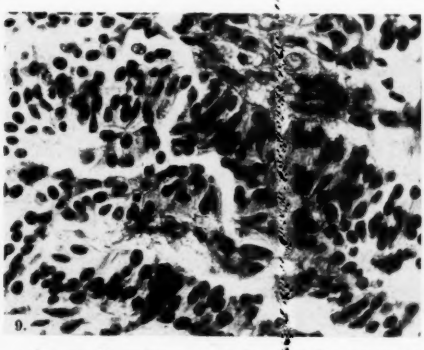
TABLE 1

Eight cases of ependymoma derived from the neurohypophysis or hypophysial stalk

Cases	Patient's Age at Onset of Symptoms	Length of Illness	Hypo-pituitarism	Sellar Changes
1 (autopsy)	16	5 yrs.	+	Slight
2	18	19 yrs.	+	None
3	19	10 yrs.	+	Enlargement
4	20	2 (11?) yrs.	+	Slight (pressure)
5	36	2 yrs.	+	Slight enlargement
6	38	13 yrs.	+	Enlargement
7 (autopsy)	45	3 mos.	probably	Slight
8	50	6 yrs.	−	Considerable enlargement

terminale. Ependymomas also vary in nature. Distinction has been made between a "tubular" form with irregularly branched cavities lined with ependymal tissue and a "solid" form in which the cells are often arranged in corona formation around the vessels. There is also a cystic form. Kernohan thought that this was a neuro-epitheliomatous form but Bailey believed that the cysts were formed around blood vessels. My ependymomas were more of the "solid" type though they had numerous cysts. As seen, however, my opinion as to their manner of development does not agree with that of either Kernohan or Bailey.

Neural tumors starting in or involving the neurohypophysis are not entirely unknown. Nonne[12] reported a case of glioma in the neurohypophysis accompanied by adiposogenital dystrophy, but unfortunately without further anatomic information. Casper[2] described a glioblastoma widely extended over the base of the brain which had infiltrated the hypophysial stalk and destroyed the neurohypophysis. He also described a bean-sized,



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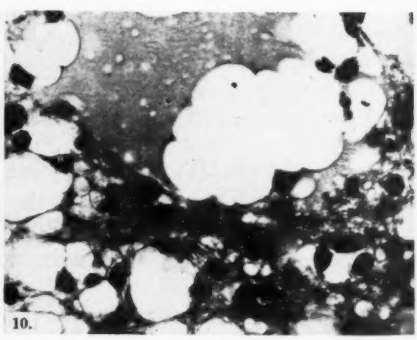

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FIG. 9. Ependymoma of the cauda equina. Heidenhain's iron-alum-hematoxylin stain.

FIG. 10. Spinal radicular neurinoma. Cyst formation through hyaline or colloid degeneration of the tissue.

entirely intrasellar ganglioneuroma (histologically diagnosed by Benda) which had destroyed the neurohypophysis. In both cases the adiposity was late in setting in and the anterior lobe appeared to be intact.

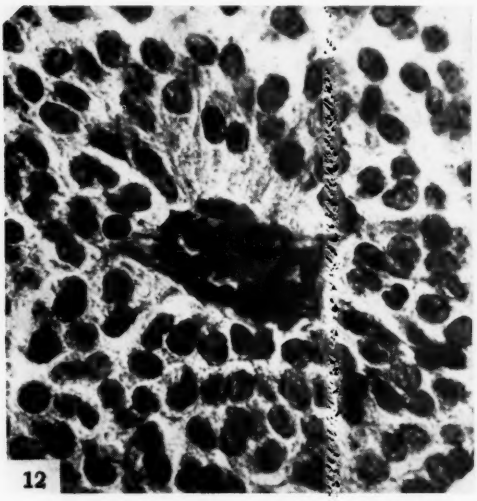
This being the case, are there any chromophobic adenomas? Yes, undoubtedly. Roussy and Oberling[16] described small chromophobic adenomas situated in the hypophysis. Close[3] reported the finding of 39 adenomas in a largely unselected series of 200 hypophyses. The majority of the tumors were found in persons over 45 years old and they were much more common in cases where benign or malignant tumors were present elsewhere in the body. However, these tumors did not resemble those of Dott and Bailey. Most of them remained small and stayed in the hypophysis; the normal acinous architecture of the organ was lost in the adenoma; only occasionally was the adenoma separated from the surrounding glandular tissue by a thin connective-tissue capsule; the stroma was poorly developed in the adenoma; the cells were smaller than the normal chromophobic variety and sometimes the tumor presented the picture of a pure "Kernhaufen." Rare examples grew to larger dimensions up to twice the size of the hypophysis, and occasionally grew beyond the limits of the sella. Two of the 39 tumors were eosinophilic and both of these were tiny growths; the others were chromophobic.

FIG. 11. *Case 6.* Hypopituitary habitus.

It may be that Roussy and Oberling's "type à petites cellules principales" represents the genuine chief cell adenoma. It is hard to say how Dott and Bailey's "simple strumous" or "alveolar" types correspond to Roussy and Oberling's types. The latter authors spoke also of a "type indéterminé." Judging by their illustrations, their type "à cellules claires" is the one which best corresponds to my ependymomas. None of these authors said anything about topographic or clinical differences shown by the various "chromophobic" types.

Those who are curious whether neurogenic hypophysial tumors may start from anatomic variants or anomalies or embryonic remains may find the following of interest. Langer[9] in 1892, Benda in 1932, Romeis[15] in 1940, Kiyono in 1926 and Floderus[5] in 1944 encountered cysts in the posterior lobe of an otherwise normal hypophysis which they believed to be remains of the infundibulum extending down in the neurohypophysis during the fetal stage, though the walls of these cysts were not lined with ependymal tissue. Judging by Langer's figures, his growths were craniopharyngiomas. Sternberg[17] reported having seen groups of large epithelial cells which he believed to be neuroepithelial elements in a hypophysial stalk and concluded it was a question of "choristoma." Priesel,[13] Kraus,[8] Roussy and Oberling[16]

and Romeis[15] observed occasional examples of "Gewebsmissbildungen" or "ortsfremde" cells of essentially similar appearance. Floderus[5] encountered the same formations and, like Sternberg, considered these palely staining, finely granulated cells with a large cytoplasm and small nucleus to be neuroepithelial and "characteristic." On the other hand, Löffler,[10] who went so far as to speak of "Geschwülste im Hinterlappen und im Stiel," regarded the cells as immigrants from Rathke's pouch. Glandular ducts and follicles with ciliary epithelium often wander into the posterior lobe from the pars intermedia or the epithelium of the hypophysial cavity. They are generally considered, therefore, as belonging to the anterior lobe. The same is true,



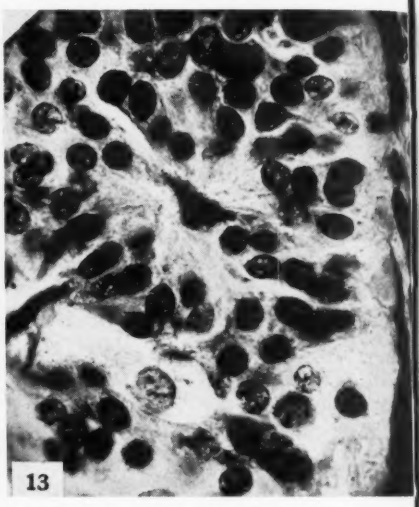


FIG. 12. Hypophysial tumor (ependymoma), partially suprasellar. Note perivascular corona formation, as in Fig. 13. Heidenhain's iron-alum-hematoxylin stain, ×700.

FIG. 13. Tumor of cauda equina (ependymoma of filum terminale). Heidenhain's iron-alum-hematoxylin stain, ×620.

Rasmussen[14] believed, of the isolated cysts lined with ciliary epithelium in the posterior lobe which Luschka described in 1860 and traced to the neural tube.

Haberfeld[6] examined the hypophysis in 6 fetuses. In 5 cases the uppermost and lowermost sections of the posterior part of the neurohypophysis showed glia-cell groups containing cysts lined with ependyma-like cells, some ciliated, the basal parts of which disappeared in a maze of glia fibrils. Haberfeld discovered similar formations in 10 out of 12 newborn children and in 1 out of 3 children under 3 years old, but never in adults. Apparently, therefore, he had observed a type of fetal formation which disappears soon after birth. Haberfeld suspected that it might be the starting-point for neoplasms.

The "fetal" cellular groups in the anterior lobe described by Kraus[8] are

particularly worthy of note. The cells correspond in type to the early embryonic hypophysial cells which occasionally (Kraus, Roussy and Oberling) proliferate to form genuine adenomas in the anterior lobe. Seen in the pictures of Kraus, and Roussy and Oberling, these adenomas are highly reminiscent of ependymoma or papilloma, though their cells are more slender. Judging from experience hitherto, this form is very rare.

It is by no means established by the present series that *all or even most of the tumors situated suprasellarly and connected with the hypophysis are ependymomas starting in the neurohypophysis or hypophysial stalk.* A much

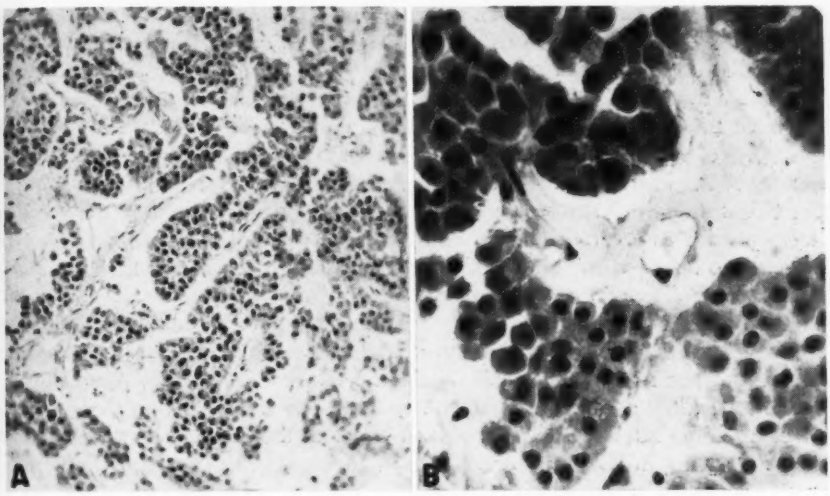


FIG. 14. Real chromophobe hypophysial adenoma, partially suprasellar. (A) Heidenhain's iron-alum-hematoxylin stain, ×170. (B) Ethyl-violet-orange stain, ×525.

more extensive analysis of a much larger series is required to settle this question. It may well be that other gliomas besides ependymomas arise in this region. The existence of genuine chromophobic hypophysial adenomas may be considered established. The only question is how big they grow and how often they occur. So much is certain, however, that this whole question must be taken up again and it may be that my assumption will prove to be correct. Several of the "chromophobic adenomas" reproduced in the articles by Dott and Bailey[4] and Roussy and Oberling[16] look to me more like ependymomas. The same is true of the drawing in Bailey's book,[1] and his description of the frequently elongated cells which "may seem to radiate around the vessels" in a scanty stroma is also suggestive of ependymoma.

Eosinophilic adenomas are of quite different nature, not only as regards staining properties. They are built up of irregularly sized, often multinuclear, generally rounded cells with irregularly sized nuclei, lying in disorderly

masses with no stroma, contain few vessels and have a marked tendency to necrosis.

A few words, finally, regarding topographic and clinical features. Bailey[1] said in 1948 that there was hardly any difference macroscopically between the two types of suprasellar tumors, viz., the chromophilic and chromophobic adenomas, to use the terminology of the Cushing school. The only exception, he said, was that the eosinophilic variety was generally smaller and grew more slowly, though it too could reach considerable size. But there was no consistent or general difference between them, in his opinion, as regards topography, as regards their direction of growth. Schaeffer (1924) has shown that the position of the chiasm in the anteroposterior direction varies greatly from person to person. My series contains cases of ependymoma the suprasellar portion of which grew so far anteriorly in relation to the chiasm as to cause blindness on one side, obviously due to pressure on the optic nerve, and temporal hemianopsia on the other. But I have also seen examples of eosinophilic adenoma with acromegaly that caused homonymous hemianopsia, in other words, that operated relatively far back in relation to the chiasm. In both the autopsied cases the suprasellar portion of the tumor lay posterior to the chiasm. On operation for suprasellar tumors it may be difficult to learn what the conditions are behind the chiasm, for the operation is generally done from the front.

As was first shown convincingly by Guthrie in 1913, Napoleon the Great suffered from severe progressive hypopituitarism. In 1932 Krogius succeeded in making the more precise diagnosis of hypophysial tumor. According to the Dott-Bailey scheme, Napoleon's case would be a textbook example of chromophobic adenoma. For my part, I am inclined to suspect an ependymoma starting in the hypophysial stalk. Judging from the clear description in Talleyrand's memoirs, Napoleon had his first observed epileptic fit in 1805, 16 years before he died. The intractable vomiting which beset him shortly before death may have been of cerebral origin. This disease must have contributed essentially to the downfall of the emperor.

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LIGATION OF THE INTERNAL CAROTID ARTERY IN THE NECK

PREVENTION OF CERTAIN COMPLICATIONS

JAMES L. POPPEN, M.D.

Department of Neurosurgery, The Lahey Clinic, Boston, Massachusetts

(Received for publication March 6, 1950)

THE sudden interruption of normal blood flow through the internal carotid arteries extracranially is recognized as a hazardous procedure in that it may result in temporary or permanent disability. The fact that many ingenious methods of ligation of the internal carotid artery have been described indicates that occlusion with a single ligature is not safe. In my experience there is no safe method of ligation. It should be executed only when an absolute emergency arises in an attempt to save life after every means known today have been utilized to determine the adequacy of the collateral circulation of the circle of Willis to carry sufficient blood supply to the hemisphere in which the direct flow will be greatly diminished by the ligation of an internal carotid artery.

Unfortunately, the circle of Willis in many instances is malformed, especially in the region of the posterior communicating arteries and, to a lesser degree, of the anterior communicating arteries. In these patients, ligation of an internal carotid artery is almost certain to be attended with a high morbidity as well as mortality, regardless of the method of ligation or occlusion.

The competency of the collateral circulation can usually be predicted with reasonable accuracy by temporary interruption of the blood flow through the internal carotid artery with digital compression. False impressions may be gathered from digital compression of an internal carotid artery. A patient with normal collateral circulation will tolerate complete occlusion by digital compression indefinitely; usually 10 minutes suffices to determine whether or not a patient can tolerate occlusion permanently. In many patients, untoward symptoms, such as dizziness, slowing of the pulse, numbness or weakness of the contralateral side or syncope, may be initiated within a few seconds or minutes after digital compression has been instituted. It is important to determine whether these symptoms are the result of ischemia from a faulty collateral circulation or of vasospasm produced by an irritable carotid sinus. The differential diagnosis can usually be determined by occluding the internal carotid artery well above or well below the carotid sinus. If similar subjective symptoms are produced by pressure well away from the carotid sinus, one must assume that the symptoms are the result of inadequacy of the collateral circulation. Another relatively satisfactory method is to interrupt the carotid sinus temporarily with local infiltration of procaine. It is difficult at times to be certain that the carotid sinus has been adequately anesthetized. If no untoward subjective symp-

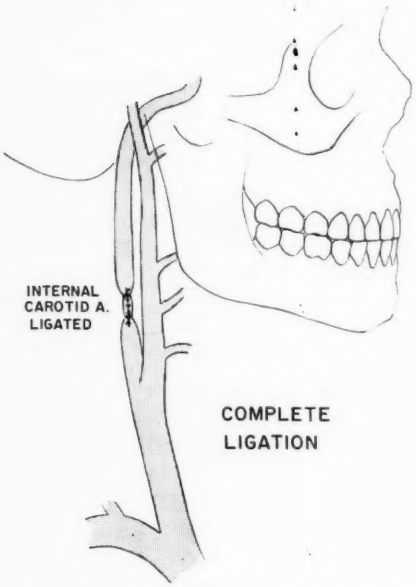
toms occur from the digital compression following infiltration of the carotid sinus with a local anesthetic agent, one is justified in assuming that an irritable carotid sinus was responsible for producing the symptoms, and not a faulty collateral circulation.

The indications for ligation of the internal carotid artery in the neck are becoming more frequent since intracranial saccular aneurysms are being visualized before death by the use of arteriograms.[3,4,5,6,7] Ligation of the internal carotid artery may be necessary in an acute emergency arising from gunshot wounds, lacerations, or erosion of the wall of an internal carotid artery by a malignant lesion or a knife wound. In these patients immediate ligation is necessary to save life, repair of the rupture being rarely possible.

In the elective ligation such as deemed necessary for intracranial lesions, the method of occlusion is highly important. I fully realize that any method utilized in a large number of patients may seem perfectly safe. However, in my previous experiences[1,2] with partial occlusion or occlusion in stages, satisfactory results were obtained in the first 40 patients, then 3 tragic delayed hemiplegias followed. Every effort should be made to prevent temporary or partial complete disability from ligation of the internal carotid artery if a patient has adequate collateral circulation which has been demonstrated to be true for a period of 36 to 48 hours, and then the patient be subjected to a sudden catastrophe due to thrombosis or embolus. It is important, therefore, when the collateral circulation has been demonstrated to be satisfactory, that every precaution is utilized to prevent these delayed tragedies. The cause of thrombosis in many instances is injury of the intima of the artery at the site of ligation. I believe that ligation of the internal carotid artery is less dangerous than ligation of the common carotid artery. The reason for this impression is that the caliber of the common carotid artery is usually 1½ to 2 times that of the internal carotid artery. I realize that theoretically the danger of ligation of the common carotid artery should be materially less since interruption of the internal carotid artery diminishes the blood flow into the ipsilateral hemisphere much more completely than does ligation of the common carotid artery (Fig. 1). With the latter the blood flow is still possible by retrograde circulation through the external carotid artery into the internal carotid artery (Fig. 2), whereas with ligation of the internal carotid artery all blood that supplies the hemisphere is interrupted from the neck, with the possible exception of the slight collateral circulation of the external carotid artery through the ophthalmic artery.

Ligation of the internal carotid artery is less dangerous than ligation of the common carotid artery because first, the caliber of the common carotid artery is greater than that of the internal carotid artery, and second, atheromatous plaques in the common carotid artery in the immediate vicinity of the carotid bulb are much more common than in the internal carotid artery. A ligature placed over an atheromatous plaque may well cause a fracture of the plaque, resulting in injury to the intima, thus developing an excellent precursor to ascending thrombosis or embolus. That

this actually happens was vividly demonstrated in a patient subjected to thyroidectomy on the general surgical service, during which procedure the common carotid artery was retracted laterally in the usual manner. The patient did not regain consciousness; when he recovered from anesthesia it was obvious that a complete contralateral hemiplegia had developed. Death occurred within 24 hours. Postmortem examination demonstrated a recent thrombosis in the entire right internal carotid artery, extending from



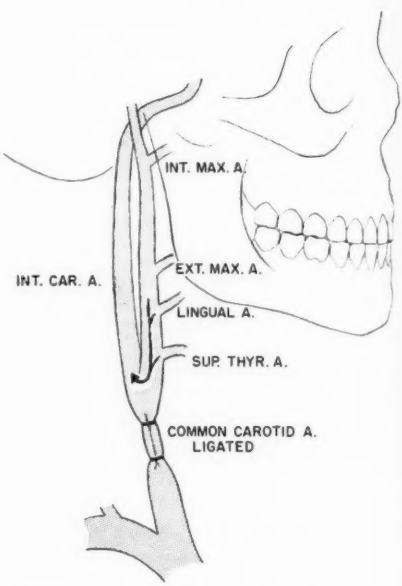


FIG. 1 (*left*). Complete ligation of internal carotid artery.
FIG. 2 (*right*). Ligation of common carotid artery with blood flow into internal carotid from external carotid artery.

a fractured atheromatous plaque in the common carotid artery just below its bifurcation at the site of retraction. It is important, therefore, during manipulation of the carotid system that extreme gentleness be used in palpating the artery; nevertheless, it should be done to prevent placing a ligature in the immediate vicinity of an atheromatous plaque.

Another instance of injury to all layers of a large vessel from ligation was demonstrated by the formation of a pulsating mass in the necks in 3 patients, 3 hours, 6 months and 12 months, respectively, following ligation. Obviously, the ligature had cut through the wall of the artery in the first instance at the site of a markedly sclerotic plaque. In the other 2 patients, aneurysms had developed at the ligature. In one patient sudden rupture of the aneurysm

took place and death occurred. The other patient was saved by excision of the aneurysmal sac in the neck.

In well over 100 ligations of the carotid system, permanent partial hemiplegias took place on the contralateral side in 4 patients. Three of these patients, however, are able to carry out their usual activities, but they do have definite residual symptoms as a result of the thrombosis.

One patient with hemiplegia on the ipsilateral side following ligation

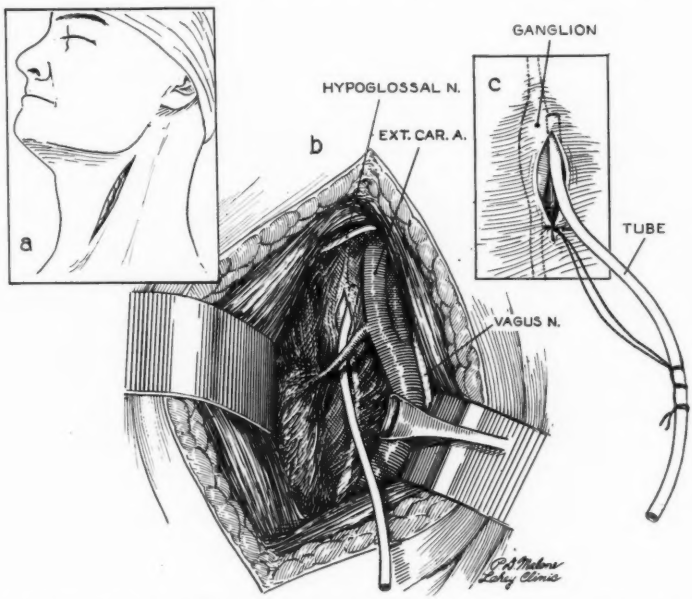


FIG. 3. (*a*) Line of incision. This is usually carried from the tip of the mastoid just anterior to the sternocleidomastoid muscle downward to a level 2 or 3 cm. below the bifurcation of the common carotid artery. (*b*) Polyethylene tubing inserted into sheath of superior cervical ganglion. (*c*) No. 00 catgut suture used to prevent displacement of tubing. At end of 3 days, the polyethylene tubing can be readily pulled out through the incision. Tubing is used as an accurate means of perfusing the ganglion continuously with a slow drip of procaine in an attempt to prevent possible vasospasm of intracranial vessels.

died as the result of rupture of an aneurysm on the opposite side. It is this episode that has made us insist that bilateral arteriograms be taken of all patients subjected to ligation of an internal carotid artery for intracranial aneurysms so that we can be certain that another saccular aneurysm is not present on the opposite side. Fortunately, in our surgical experience at the clinic with 152 cases of intracranial aneurysms, 3 per cent of the aneurysms were bilateral and 7 per cent multiple but unilateral.

The following technic was utilized for 50 patients who have survived for more than 3 years.

The anesthesia is of especial importance. It is necessary that the patient is cooperative, and therefore, regional or local anesthesia is preferable. After adequate premedication, most emotional patients can be managed if they are made aware of the importance of their cooperation. Many patients become very restless if the drapes are applied so that proper oxygenation is not possible. Adequate air or oxygen is imperative to prevent building up of carbon dioxide under the sterile drapes.

An oblique incision is made anterior to the sternocleidomastoid muscle, starting immediately below the tip of the mastoid process and carrying it down below the level of the bifurcation of the carotid artery (Fig. 3 *a*). The carotid sheath is allowed to remain in contact with the carotid arteries, dissecting the internal jugular vein and the vagus nerve from the sheath. It is usually necessary to ligate the common facial vein as well as the lingual and the superior thyroid veins to allow adequate exposure of the internal carotid artery and the carotid bulb. The carotid sinus is anesthetized by infiltrating a small amount of procaine directly into it. The external carotid artery is separated from the internal carotid artery by incising the adventitial layer and separating it from the lateral wall of the external carotid. This allows the full thickness of the adventitia to be utilized in forming a natural band for constricting the internal carotid artery. The lumen of the internal carotid artery is then obliterated by interrupted black silk sutures, as shown in Fig. 4 *a*, *b* and *c*. Three layers usually suffice to occlude the lumen completely. It is, of course, obvious that the adventitial layer will stretch as time goes on and allow the lumen to reopen. I have no factual evidence as to the length of time this would take. To prevent this, however, two large black silk sutures are placed, one on the extreme cephalad end and the other on the extreme caudad end of the occlusion, usually 3 to 3.5 cm. apart (Fig. 4 *e*). These ligatures are tied snugly but not so tight as to injure the intima. At the conclusion of the ligation the sheath over the superior cervical ganglion is opened to allow a small polyethylene tubing to be inserted so that a constant drip of procaine may perfuse the ganglion for 2 or 3 days following the operation (Fig. 3 *b* and *c*). The tubing is kept in place with plain, No. 2 or 3 catgut sutures so that the tubing can be readily removed at the end of 3 days. This is done with the hope that it will prevent vasospasm and allow maximum collateral circulation during the crucial time following ligation.

The patient is in the horizontal position throughout the operation so as to maintain a normal blood pressure during and following the operation. Careful hemostasis is instituted so that heparin may be given if necessary following operation. It had been my opinion that heparin should be given 4 hours postoperatively and continued for 3 or 4 days. It was necessary to modify this opinion following an experience in which heparin was given in a case in which the aneurysm had recently ruptured. Several hours after administration of the heparin a fatal subarachnoid hemorrhage took place. Heparin is given to all patients 4 to 6 hours following ligation who have not

had a subarachnoid hemorrhage within 3 weeks previous to the ligation and also to all patients who have large, inoperable intracranial aneurysms and who have shown no signs of subarachnoid hemorrhage but who have local neurologic changes.

The blood pressure must be carefully watched throughout the operation and following the operation. There is very little question that ligation performed at the time of a lowered blood pressure is conducive to serious sequelae. The patient is returned to the ward with the bed in shock position and the patient is kept in an oxygen tent 12 to 24 hours. Proper oxygenation

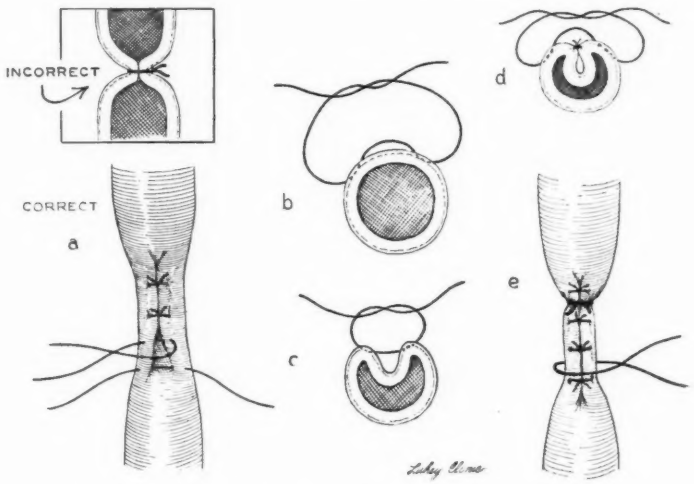


FIG. 4. Imbrication of the large vessel by utilizing the adventitial layer.

is worth while in a patient whose rate of circulation is greatly lowered as a result of the ligation.

The operating room is kept prepared for the immediate removal of the ligatures if untoward signs are noted in the first 4 hours. If hemiplegia occurs after 4 hours, removal of the ligatures has not been beneficial. Symptoms from ischemia as a result of occlusion usually manifest themselves well within that time.

Following operation the site of ligation in the neck must be carefully handled. No attempt should be made to palpate the neck since, if thrombosis has occurred at the site of ligation, a thrombus may be dislodged.

Thrombosis and embolus formation probably occur less frequently in cases of ligation of the internal carotid artery in which immediate division of the artery is carried out rather than ligation in continuity. There is no recourse, however, if hemiplegia occurs in 2 to 3 hours following ligation, as happens in a few cases.

Ligation of the internal carotid artery in a patient in shock will increase both the mortality and the morbidity. The blood pressure should be elevated by transfusions if ligation becomes necessary as a life-saving mechanism in an actively leaking aneurysm. An actual "blow-out" of an intracranial aneurysmal wall will result in death so quickly that operation is out of the question unless by pure chance the rupture occurred as the internal carotid artery was exposed immediately prior to ligation. It is doubtful that ligation at such a time would save the patient's life.

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DIVERGENCE PARALYSIS WITH INCREASED INTRACRANIAL PRESSURE

MAX CHAMLIN, M.D., AND LEO M. DAVIDOFF, M.D.
Neurosurgical Service, Beth Israel Hospital, New York City

(Received for publication March 23, 1950)

DIVERGENCE paralysis was first described by Parinaud in 1883. Since then, it has been observed by other investigators and there seems to be some good evidence that a center for divergence exists in the midline of the brain stem, probably between the nuclei of the 6th nerves. The latter view was advanced by Bruce[5] in 1935.

However, divergence paralysis is still rather infrequently described in the neuro-ophthalmological literature. While this is undoubtedly due to a low incidence of occurrence, it is also possible that it is occasionally not recognized clinically, but passed off as atypical diplopia with no individual ocular muscles being found at fault. However, despite this apparent infrequency, we encountered 3 cases of divergence paralysis on our service over a period of 3 months, although we are not aware of having seen divergence paralysis for at least 4 years prior to this period.

The syndrome of divergence paralysis is described by Duke-Elder[7] as "the appearance of a convergent strabismus with homonymous diplopia of the concomitant type when the eyes view a distant object, together with the absence of any limitation of movement of either eye in ductions or in versions in any part of the field." Normally, when one changes his gaze from a near to a more distant object, one or both globes may turn out so that the apex of the angle formed by the visual axes of the two globes is transferred from the near object to the more distant one. This action is called divergence, and, to a great extent, invokes the action of the external recti muscles. In divergence paralysis, the eyes can converge normally, and, by relaxing this convergence, can view an object singly up to a certain near point which is usually 10 or 20 inches in front of the patient. Beyond this near point, divergence power is necessary for binocular single vision, and, since divergence is absent, the patient begins to see double. The further away from this near point the object is held, the greater the distance between the two images. At the same time, while the disconjugate movement of divergence is gone, the individual ocular muscles and conjugate movements show no palsies. In other words, the abduction and adduction, as well as dextro- and laevoversions are normal.

At any one distance from the patient, the distance between the two images is, with some minor variations, constant, in all the cardinal directions of gaze. This is in contrast to the picture in an individual muscle palsy, where the separation of images is greatly increased in the field of action of the paretic muscle.

In making a differential diagnosis, two conditions may cause confusion—

convergence spasm and bilateral 6th nerve palsy. The latter can be ruled out by the lack of external rotation of each eye on attempted laevo- and dextroversion. In convergence spasm, as fixation approaches the near point the diplopia increases, while, in divergence paralysis, it decreases.

While divergence paralysis was described as possibly due to a functional disturbance by von Hippel[8] and Clark,[6] Savitsky and Madonick[13] pointed out that among over 2000 cases of psychoneuroses observed in private practice, no case of divergence paralysis was encountered that could be considered entirely functional.

In organic disease, divergence paralysis has been described in the following conditions:

(1) Inflammatory or toxic diseases of the cerebrum. Duke-Elder[7] cited a number of references where divergence paralysis occurred in syphilis and tabes, encephalitis, multiple sclerosis, diphtheria, poliomyelitis, influenza, chorea and lead poisoning.

(2) Cerebral hemorrhage. Alger[1] and Wheeler (cited by Duke-Elder,[7] p. 4176) reported 1 case each in which they assumed the divergence paralysis was due to hemorrhage into the divergence center, because of the rather sudden onset of symptoms.

(3) Head trauma. Divergence paralysis was reported by Bielschowsky,[3] Weed,[16] and Savitsky and Madonick,[13] the latter expressing the opinion that in such cases there was trauma to the hypothetic center for divergence in the midbrain.

(4) Brain tumor. There are 7 cases in the literature in which the nature and site of the lesion were confirmed either by necropsy or surgery, as well as 3 cases in which the same picture seemed to be present, but was unverified. The latter 3 cases were reported by Straub,[15] Holden,[9] and Howard.[10]

The 7 verified cases were reported from 4 different sources. Bender and Savitsky[2] reported a case of a small vascular tumor in the pons verified by necropsy. Divergence paralysis was the chief complaint during life and papilledema was present.

Lippmann[11] described a case of cerebellar tumor verified by necropsy. Divergence paralysis had been present for 2 years prior to death and papilledema developed before death.

Robbins[12] reported a case of divergence paralysis in which the patient died after 6 months. Necropsy revealed a cerebellar cyst. While papilledema was not observed during life, the ventricles were found to be dilated.

Savitsky and Madonick[14] reported 4 cases of posterior fossa tumor with divergence paralysis, all verified by surgery. Two were acoustic neuromas and the other 2, cerebellar tumors. All patients showed papilledema preoperatively. In 3 cases, the tumor was removed and the divergence paralysis cleared completely as the papilledema diminished. In the 4th case, due to the vascularity of the tumor, only a biopsy was taken, and the divergence paralysis and papilledema were still present at the time of their report. They

had not found divergence paralysis in any cases of increased intracranial pressure without tumor and concluded that divergence paralysis is due to an involvement of the divergence center in the brain stem in some way.

AUTHORS' CASES

We are reporting upon 3 cases, each of which showed evidence of increased intracranial pressure, and typical divergence paralysis. All 3 patients were operated on, with disappearance of the increased intracranial pressure and complete disappearance of the divergence paralysis.

Case 1. B. N., a 16-year-old girl, complained of dizziness for 3 months and double vision for 3 weeks. There was typical divergence paralysis with a separation of images starting at 13 in., where the patient reported a 3-in. separation took place. At 7½ ft., the images were about 15 in. apart. Individual duction and version actions of all the extraocular muscles were normal. The fundi showed congestion of the nasal margins of the discs, and the blind spots were enlarged to a little beyond the limits of normal (with 2/2000 white, OD measured 8½° by 10°, and OS, 8½° by 9½°).

A ventriculogram revealed internal hydrocephalus with evidence of a tumor in the posterior fossa. A craniotomy was performed and a right cerebellar hemangioblastoma removed.

One week postoperatively, with recession of the papilledema, the divergence paralysis had cleared considerably and, in 10 days, it was all gone. At the time of this report, 8 months later, there was no recurrence of any symptoms.

Case 2. S. B., a 56-year-old man with a 6-month history of headache, complained of seeing double for the past 2 weeks. Examination revealed bilateral frank papilledema and typical divergence paralysis. The patient reported a separation of images starting at 6 in. and increasing with the distance from him until, at 20 ft., he reported about 36 in. of separation. Individual muscle action of the internal and external recti was normal, so that both left and right conjugate gaze were unimpaired.

Operation revealed subdural hematomas bilaterally. These were evacuated and the patient was relieved of headache.

Five days postoperatively, the separation of images at 20 ft. was reduced from 36 in. to 10 in. On discharge 15 days after operation, the diplopia was entirely gone and the papilledema had receded. At the time of this report, 5 months postoperatively, there has been no recurrence of symptoms.

Case 3. P. T., a 35-year-old man, presented himself with a history of headache for 10 days. The disc margins were blurred nasally and the blind spots were definitely enlarged. With 2/2000 white, the right blind spot measured 8° by 13° and the left, 10° by 12°.

Ventriculograms were made and all the ventricles filled well. There were no clear-cut deformities or obstruction to warrant any surgical procedure at that time and he was sent home.

He returned 19 days later with a history of increasing headache as well as the onset of diplopia soon after having left the hospital. Examination revealed frank

papilledema bilaterally, as well as divergence paralysis. At 4 ft., he reported a separation of 10 in. of images and, at 15 ft., about $4\frac{1}{2}$ ft. of separation. On the other hand, abduction and adduction, as well as left and right conjugate gaze, were normal, so that none of the individual recti muscles was paretic.

Following a subtemporal decompression, the papilledema and diplopia subsided. The final diagnosis was serous meningitis. Nine days postoperatively, the diplopia had diminished so that, at 15 ft., the separation was only 2 ft. Three weeks after operation the papilledema was almost all gone and no diplopia was present.

COMMENT

In all 3 of our cases of divergence paralysis, the common accompanying factor was increased intracranial pressure. Only in Case 1 was there a local space-occupying lesion in the posterior fossa. In Case 2 the increased intracranial pressure was due to bilateral supratentorial subdural hematomas. In Case 3 it was associated with serous meningitis.

In the 7 cases of proven brain tumor reported by other authors, all but 1 showed papilledema, and that one showed dilated ventricles at necropsy, thus indicating obstructive hydrocephalus and therefore probably increased intracranial pressure. In addition, in all these 7 cases there was a local space-occupying lesion in the posterior fossa.

The questions that arise are whether posterior fossa lesions near the region of the so-called divergence center plus increased intracranial pressure are necessary to produce divergence paralysis, or whether increased intracranial pressure alone can produce it. Our Cases 2 and 3 certainly showed no evidence of local lesions in the posterior fossa and the divergence paralysis cleared with the subsidence of papilledema.

While in some of the previously reported 7 cases diplopia had been present for even 2 years prior to operation or necropsy, in some instances of spontaneous remissions it would be difficult for us to prove that there was not a low-grade increased intracranial pressure during that time. Certainly, the absence of visible papilledema does not rule out increased intracranial pressure.

In view of these facts, and the occurrence of divergence paralysis in 2 of our patients with increased intracranial pressure but no evidence of a local space-occupying lesion in the posterior fossa, it seems reasonable to assume that increased intracranial pressure alone may cause divergence paralysis. The occurrence of 6th nerve palsy with increased intracranial pressure is well known. Whether the mode of action is similar in the two conditions remains to be seen. As a matter of fact, Bielschowsky[4] observed cases of typical divergence paralysis that went on to true abducens palsy. He considered this as evidence of an organic lesion near the intact abducens nucleus and later extending to and finally involving the nucleus itself. He considered this as good evidence that true divergence innervation exists.

From a clinical point of view, we think that divergence paralysis is seen as an infrequent accompaniment of increased intracranial pressure, but must not be regarded as pathognomonic of a lesion in the posterior fossa. In other

words, increased intracranial pressure alone seems to be capable of producing divergence paralysis. The additional presence of a lesion in the region of the so-called divergence center does not seem to be necessary for its production.

CONCLUSIONS

1. Divergence paralysis, while infrequently encountered, must be thought of and looked for in cases of increased intracranial pressure, and should certainly be looked for in all cases of diplopia with no apparent limitation of individual ocular muscle action.

2. Divergence paralysis is found in cases of increased intracranial pressure, and, while frequently associated with a space-occupying lesion in the region of the so-called divergence center, has been demonstrated in 2 cases without any clinical evidence of any such posterior fossa lesion.

3. Divergence paralysis must not be considered pathognomonic of a local lesion in the posterior fossa.

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UNUSUAL CONGENITAL ANOMALIES OF NEUROSURGICAL INTEREST IN INFANTS AND CHILDREN*

EDGAR A. KAHN, M.D., AND LLOYD J. LEMMEN, M.D.
Department of Surgery, Section of Neurological Surgery, University Hospital
Ann Arbor, Michigan

(Received for publication April 11, 1950)

THIS report is based upon some of the rarer congenital anomalies encountered in the Neurosurgery Clinic at the University Hospital during the past few years. No attempt has been made to review the literature on this subject as this has been done so thoroughly by Ingraham and his associates.[3] We wish rather to discuss the practical solution to some of the problems one faces in dealing with these unusual congenital malformations.

Case 1. B. W. was referred on July 5, 1949 to the University Hospital by Dr. R. Atchison of Northville, Michigan, 9 hours after delivery, because of a large congenital defect of the scalp. The pregnancy had been normal, but the description of the delivery is most interesting. Dr. Atchison was told by the nurse that the membranes had ruptured. It was a vertex presentation and on seeing only the shiny scalp defect, it was naturally thought that the membranes were still unruptured. He picked up a hemostat and was about to open the membranes, when fortunately at that moment the patient had a severe labor pain and with this further bulging he realized that it was the child's head presenting. The remainder of the delivery was normal.

The family history records that a sibling with an entirely similar scalp defect, but no other recognizable congenital defects, died of meningitis at 12 days. This sibling had been seen by us 3 days post partum but the condition was unlike anything we had encountered previously and we deemed the lesion inoperable. The parents were not related to each other and there was no history of a similar lesion in either family.

Examination. A scalp defect, 5×7 cm., extended between the anterior and posterior fontanelles, in which there was no normal skin. The defect was covered only with a shiny, thin transparent membrane beneath which could be seen large venous spaces. It was well demarcated from the normal scalp. Palpation gave the impression of incomplete closure of the skull over the midline at the center of the defect (Fig. 1A). There was also present a deformity of one finger and both feet in that some of the distal phalanges were absent. Several hours after admission, the membrane became dry and lost its sheen.

X-ray films showed the anterior fontanelle to be within normal limits. The posterior fontanelle was difficult to make out, but there was no obvious defect in the calvarium between the two fontanelles as had been suspected on palpation.

Operation. As soon as studies were completed, the scalp defect was excised under local infiltration with metycaine ½ per cent. No abnormality was found in the underlying bone. After mobilization of the scalp bilaterally down to the ears

* Presented at meeting of the Harvey Cushing Society in Colorado Springs, Colorado, April 17, 1950.

and by means of rotation flaps, the entire defect was closed primarily (Fig. 1B). Convalescence was uneventful.

Histological Examination (Dr. E. Cawley). "As the defect is approached, the epidermis becomes thinned and gradually fades out, exposing markedly atrophic corium approximately one-third the thickness of normal corium. There are no sebaceous or sweat glands. A striking feature seen in the atrophic portion is calcification of the hair follicles. Diagnosis: Congenital ectodermal defect of the scalp."

Comment. Congenital defects of the scalp are extremely rare, only 76 cases having been described up until 1930. The majority of these were in

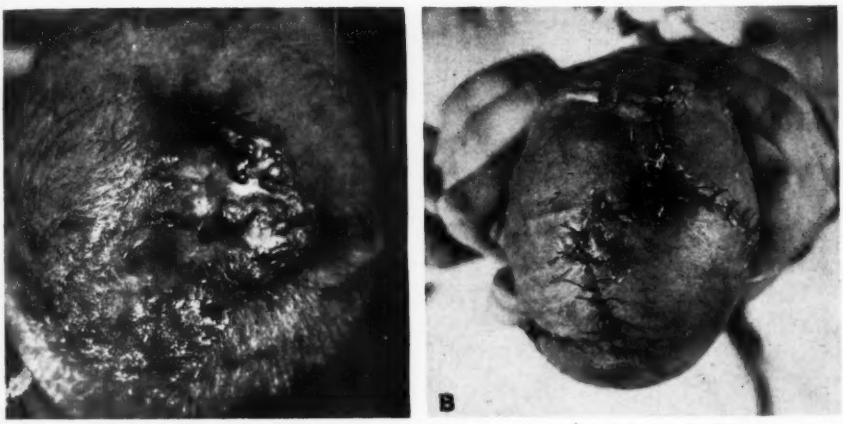

FIG. 1. *Case 1.* Pre- and postoperative photographs of congenital scalp defect.

the same location. A search of the literature revealed no case subjected to surgery. Individuals with large defects have died as a rule of meningitis or hemorrhage from the sagittal sinus or the large veins beneath the defect.[7] It should be stressed that these large congenital scalp defects should be operated upon as an emergency procedure. Procrastination will lead to infection making a plastic procedure most difficult.

Case 2. D. C., a 3-month-old white boy, was admitted on Jan. 8, 1947, because of a mass extending from the bridge of his nose (Fig. 2A). This appeared to have increased somewhat in size since birth and fluctuated when the infant cried. The mass, covered by normal skin, was walnut-sized and extended into the palpebral fissure of the right eye and almost into that of the left. A bony defect of the skull was palpable around the periphery of the mass. Neurological findings were entirely normal.

X-rays of the skull showed a spherical soft tissue mass presenting through a defect involving the medial halves of both supraorbital ridges and the inferior frontal area, as well as the expected position of the superior nasal bones.

Operation. On Jan. 13, 1947 the sac of the encephalocele was circumscribed superiorly by an incision down to the periosteum. The incision was then carried down to the root of the nose where the scalp and skin of the face were dissected from

the sac until the entire defect was exposed. The bony defect was larger than a 25-cent piece. The sac, which contained fluid and heterotopic brain, was opened. This brain substance was excised until fairly healthy cortex was encountered. This meant excising the tips of both frontal poles, following which the brain lay free within the cranial cavity. The crista galli was seen. Dural flaps were fashioned and a good closure was obtained. A flap of periosteum was swung over the defect leaving a pedicle for blood supply. The scalp and skin of the face were sutured in more or less of a triangular fashion with one layer of silk (Fig. 2B).

Histologically there were multiple islands of neural tissue with some glial elements. Some of the tissues showed organization suggestive of cortex.

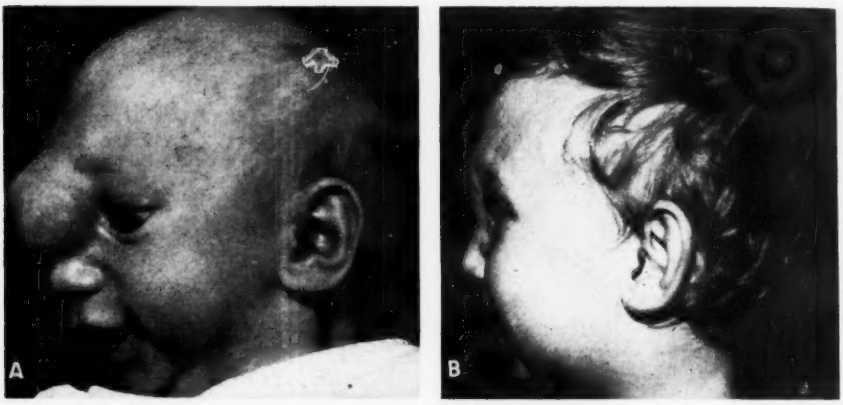

FIG. 2. *Case 2.* Pre- and postoperative photographs of anterior encephalocele.

Course. In the past 3 years the patient has done well except for several convulsive episodes. Grossly the defect is filled by solid bone which is confirmed by x-ray. His intelligence is normal.

Case 3. S. M., a newborn baby, was admitted on April 1, 1946 because of a huge mass in the interscapular region noted at the time of delivery (Fig. 3). This mass was 5×10 cm., with its long axis in the long axis of the body. Huge veins beneath the skin that covered the cystic mass were a striking feature. On deep palpation firm tissue was present. There was no neurological abnormality.

X-ray films of the chest and spine showed a large mass extending posteriorly from the mid-dorsal spine. There was marked widening of the neural canal in the area of the origin of the broad pedicle of the mass.

Operation. On April 19, 1946 under local anesthesia, an incision was made around the lower part of the base of the mass, where the large veins were encountered. There was then entered a cavity containing a large amount of bloody fluid. The mass extended laterally from a defect in the vertebrae which was about 2.5×1 cm. There was no dural sac surrounding this vertebral defect as in the ordinary myelomeningocele. Some fluid appeared in the defect but its origin could not be determined. The entire mass was removed down to what was thought to be epidural space, but the anatomy was poorly defined. The remaining tissue lay free in the

vertebral defect. A fascial closure could not be obtained. Muscle flaps and a layer of subcutaneous tissue were sutured over the defect. The skin was closed in a triangular manner, that on the right being exceedingly thin.

Course. The child, now 4 years old, is stated by the family physician to be normal in all respects.

Pathological Examination. Section of the mass removed revealed two large cystic spaces which occupied the center except for a rim of tissue about 1 cm. in thickness beneath the surface epithelium. On microscopic study this area was found to be composed predominantly of aberrant brain with embryonic neurons and neuroglial elements. There were numerous vascular channels in and surrounding

FIG. 3. *Case 3.* Preoperative photograph of dorsal teratoma.

the aberrant brain tissue. Cartilage, mucous glands, nerve fibers, ganglia, and mucosa, which in one area resembles respiratory type epithelium, and in another area intestinal type mucosa, are present in only a limited area. In the deeper sections there was ependymal lining with areas of choroid plexus. Diagnosis: Trigerminal teratoma.

Comment. This is an unusual case in that the sac one ordinarily sees in a myelomeningocele arising from the vertebral defect was absent grossly and histologically. The differential diagnosis between teratoma and myelomeningocele was established in this case only by a detailed histological examination of the entire specimen. This midline mass probably prevented the normal bony closure, thus producing the vertebral defect.

Case 4. S. T., a 2-year-old boy, was admitted on Oct. 10, 1949 because he had had meningitis secondary to a congenital dermal sinus infection in the lumbar region. One month prior to admission a small papule developed in this area which

discharged pus. On Sept. 26, 1949 muscle twitchings, stiff neck and fever developed. Lumbar puncture revealed a cell count of 13,900/c.mm., 99 per cent of which were polymorphonuclear leukocytes. The culture, however, was negative.

The patient was treated for meningitis by Dr. M. Cooperstock at Northern Michigan Children's Clinic with sulfadiazine, streptomycin and penicillin to which he readily responded. As soon as the symptoms of meningitis had subsided, he was referred to the University Hospital.

Examination. There was an erythematous, pigmented area of skin over the level of L4, at the center of which was a dimple surrounded by a tuft of hair. One cm. to the right of the dimple was a small opening which exuded thick yellow pus.

FIG. 4. *Case 4.* Surgical specimen showing dermal sinus leading to yellow nubbin (see Fig. 5 for details of fields A and B).

Operation. On Oct. 13, 1949, under avertin and ether anesthesia, a horizontal elliptical incision was made excising this area of pigmentation. The dissection was carried down to the deep fascia and a stalk, 2 mm. in diameter, was isolated. The muscles were then stripped from the spine and lamina of a normal vertebra above. This lamina was removed and normal epidural fat showing no signs of infection was uncovered. Beneath this was normal dura. Bone was removed until the point was exposed where the stalk entered the posterior dura. It could then be seen that this stalk was running from the skin directly to the dura and entering it at an acute angle. The dura was then opened longitudinally without entering the arachnoid, excising the stalk with a narrow dural cuff. Attached to the stalk intradurally and adherent to a vascular arachnoid over a nerve root was a small nubbin of tissue, 3×2×1 mm., which was firm and yellowish in color (yellow nubbin). This was excised along with the stalk as a single specimen. The other nerve roots of the cauda equina appeared normal and there was a free flow of colorless CSF. The dura was readily closed.

Course. This was entirely uneventful and a 4-month postoperative check-up by Dr. Cooperstock showed no neurological defect.

Microscopic Examination. From the skin a cornifying stratified squamous epithelial tract can be traced in serial sections (Fig. 4), through the corium down to a blind end (yellow nubbin) lined by stratified ciliated columnar epithelium with a definite basement membrane (Fig. 5). Some of the cells have the appearance of goblet cells seen in respiratory epithelium. There are small mucous glands, inflammatory cells, foreign body giant cells, and a solitary nerve rootlet in the tissue surrounding the blind end. Throughout the sinus tract may be seen keratohyaline and amorphous debris and in the blind end (yellow nubbin) there are necrotic pus cells. Diagnosis: Teratoid.

Comment. The histological structure of the nubbin is very similar to two larger lesions described by Rubie and Fulton[4] and others. Had not infection intervened, we have the potential development, depending upon which process would predominate, of either a cholesteatoma from the kerato-

hyaline from the stratified squamous epithelium or a mucous cyst from the mucous glands. Adams and Wegner[1] collected 6 cases of the latter which caused intermittent compression of the spinal cord. Sachs and Horrax[9] found 14 reported cases of intraspinal epidermoids and dermoids associated with dermal sinuses. They found no teratoid lesions in the cases they collected of this type.

Case 5. D. G., a 9-day-old white baby girl, was admitted on April 1, 1949 because the repair of a myelomeningocele noted at birth had not held. She was born following

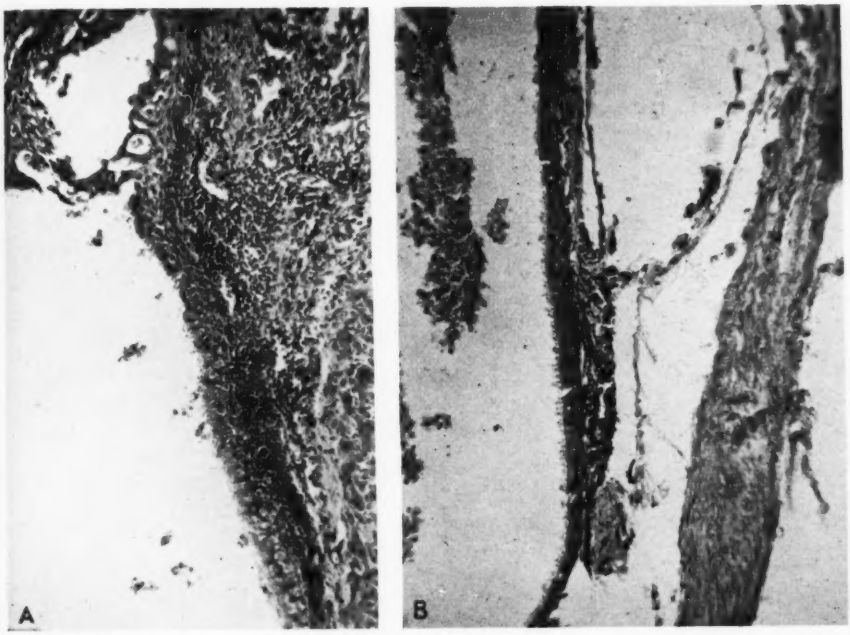

FIG. 5. *Case 4.* Photomicrographs of fields marked in Fig. 4. (A) Junction of stalk and yellow nubbin with transition of stratified squamous epithelium to ciliated columnar epithelium (hematoxylin eosin, ×120). (B) Yellow nubbin attached to nerve root. Pus cells are shown in the cavity lined by ciliated columnar epithelium (hematoxylin eosin, ×120).

a normal pregnancy and delivery. Because the membrane was so very thin and it appeared that rupture was imminent the family physician closed skin and fascia over it without opening the sac. A wound dehiscence occurred on the 8th day and the dural sac herniated through the open wound without rupturing.

Examination. The entire operative incision was open and what appeared to be a purulent dural sac was protruding through the opening. There was a mild temperature elevation which was felt to be secondary to the infected wound. There was no weakness of the lower extremities and the anal sphincter was intact.

Course. The baby was placed in Trendelenburg position and furacin dressings

were applied to the wound. She was given full therapeutic doses of penicillin and sulfadiazine. After 8 days the fever subsided and the gross evidence of infection around the postoperative wound had cleared.

Operation. On April 11, 1949, under ether anesthesia, the granulating meningocele sac was circumscribed into fairly normal skin. The dissection was carried down to the base of the sac, the neck of which measured 1×0.5 cm. as it entered the bony defect. The sac was opened and the abnormal spinal cord tissue which was adherent to it was dissected free so that the spinal cord lay free in the dural canal.

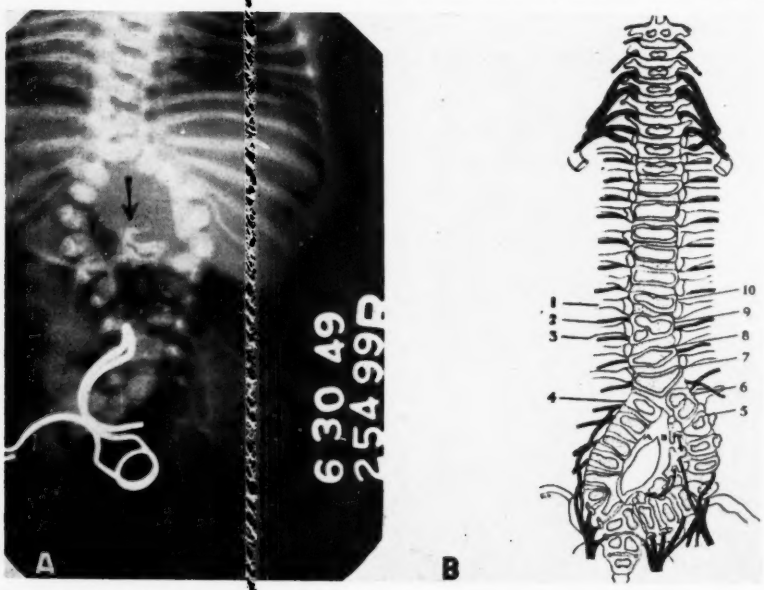

FIG. 6. *Case 6.* (A) Roentgenogram. Arrow points to bony spicule found at operation between the two dural sacs. (B) Drawing of dissected autopsy specimen from case of diplomyelia described by Saunders,[10] showing marked similarity.

A good dural closure was obtained using fine chromic catgut. Fascial flaps were sutured over the vertebral defect, giving an excellent closure.

Course. The wound healed by primary intention. Examination 1 year postoperatively showed the child to be normal in all respects.

Histological Examination. There was a marked infiltration of polymorphonuclear cells, plasma cells, and foreign body giant cells in the connective tissue, adipose tissue and aberrant nervous tissue. There were colonies of microorganisms in the more superficial areas of the skin.

Comment. This case shows that with the present day antibiotics an extensive dissection of this type can be carried out in the presence of controlled infection.

Case 6. M. L., a 4-month-old girl, was admitted on Nov. 7, 1949, with a mass in the lumbar region present since birth. The mass, well covered with skin, was

9×6×2 cm. and pulsated on crying. There was slight weakness of the left lower extremity and the foot was definitely smaller than the right.

X-ray films of the spine showed an extensive separation of the two halves of the vertebral bodies beginning at D8 and extending throughout the lumbar area, with the hemi-vertebrae widely separated in the midportion. At the level of L2 there was a spicule of bone extending posteriorly into the soft tissue (Fig. 6A). (This spicule of bone was absent on the postoperative films.)

Operation. On Nov. 14, 1949, under ether anesthesia, an elliptical transverse incision was made around the mass. A normal spinous process and lamina was exposed above this mass and the dissection was then carried down to where a lipomatous mass was seen coming out of a bony defect. Further dissection revealed a bony mass projecting posteriorly from the anterior part of the bony canal and this was splitting the dural sac. The area of doubling of the spinal cord around the bony mass was about an inch in length; above and below this point the dural sac appeared normal. The dura on the right was firm and to all appearances normal and therefore was not opened. The lipomatous mass was coming only from the dural sac on the left. On dissecting this further, nerve roots could be seen coming from a large mass of rudimentary cord substance infiltrated by fat. Just above the midline bony mass there was a midline dehiscence covered by a soft fluctuant mass which undoubtedly was retroperitoneal tissue. The bony mass was then excised so that it would no longer keep the main dural sac impaled upon it. CSF came only from the area of the lipoma of the cord, which was partially excised. It was impossible to get good fascial flaps, only muscle flaps and subcutaneous tissue being available for closure.

Postoperatively her course was uneventful except that it was necessary to aspirate CSF which collected beneath the wound for about 2 weeks. Four months after operation the involved foot appeared to have normal strength. The child, now 8 months old, is beginning to stand.

Comment. Diplomyelia or diastematomyelia is a term applied to apparent doubling of the spinal cord. The x-ray picture of this condition was recently pointed out to us by Dr. E. B. Neuhauser.[5] Herren and Edwards[2] in a review of the literature found 42 cases in which there was a doubling of the cord, which they believed was due to partial twinning. They found that it occurred in all areas of the cord and was found at all ages of life. Diplomyelia may in rare instances be present unassociated with a bony defect. In a quarter of the cases there was an osseous or cartilaginous mass in the midline separating the two cords. They suggested that this is also a manifestation of partial twinning. Diplomyelia becomes of surgical importance only when mesodermal derivatives, such as bone, ligaments, or cartilage, become secondarily involved in the lesion and anchor the cord, producing a mechanical hindrance to its ascent. This case also shows a marked similarity to the case reported by Saunders[10] in which a dorsal intestinal herniation associated with diplomyelia was verified by autopsy (Fig. 6B).

This is the third case of this type that we have confirmed by operation in the past 2 years. Pickles[8] in a review of the literature found the total number of reported cases including his own to be 47; 6 had been found at operation and 5 of these patients survived.

Case 7. A. K., a 6-day-old girl, was admitted on Dec. 6, 1947 because of a large mass over the sacral region extending from the sacral region through the posterior part of the perineum (Fig. 7A). It was noted that the coccyx could be felt fairly distinctly giving the impression that it was being pushed backward. The mass was 30 cm. in circumference and did not transilluminate. The anus was situated at the anterior margin of the mass, being drawn up to some extent into its wall. The anus was patulous and appeared to have lost its tone.

X-ray films of the spine and pelvis demonstrated a large soft tissue mass. There has been noted, however, in retrospect a small gas shadow within the mass, which must represent intestine displaced downward and laterally (Fig. 7B).

Operation. On Dec. 8, 1947, under ether anesthesia, a curved incision was made

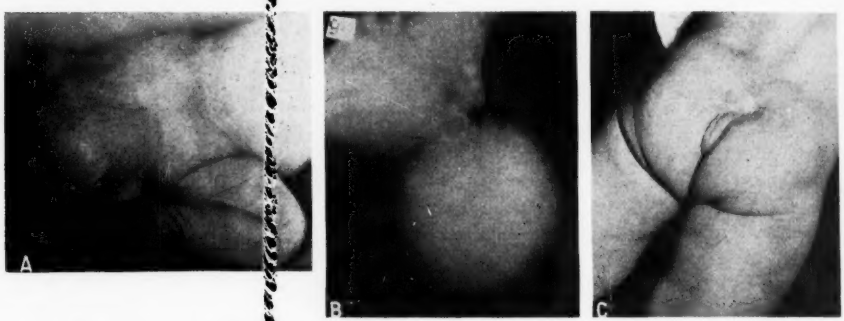

FIG. 7. *Case 7.* (A) Preoperative photograph. (B) Roentgenogram showing gas shadow extending into mass. (C) Photograph 1 year postoperative.

over the sacral swelling. A large cyst containing bloody fluid was entered and it was believed that this must be the sac of a myelomeningocele. The base of the sac was dissected free and was exceedingly wide. Another sac was now entered but no nervous tissue was present. The coccyx and sacrum were pushed upward by the swelling. A mass about 4 cm. in diameter, covered with tissue that looked like peritoneum, was then seen. The mass bulged backward markedly with every respiration and it was still thought to be a myelomeningocele. This sac was opened and intestine could now be seen. It was then realized that this was a posterior herniation which contained lower colon and small intestine. There were three heavy layers of fibrous tissue which had been dissected free which were used to repair the hernia.

The baby's *postoperative course* was uneventful and when seen a year later the wound was solid with no pulsation on straining. She was beginning to walk and had normal bowel movements. The anal sphincter was intact (Fig. 7C). Recent checkup examination by Dr. Cooperstock 2 years after operation reveals the patient to be normal in every way.

Histological Examination. The specimen was a trigerminal teratoma with spaces lined by a respiratory type of epithelium, glial tissue, glands and squamous epithelium.

Comment. This was a most unusual condition and we had never encountered it before. The only vertebral defect was the pushing upward and backward of the sacrum and coccyx. The large posterior hernia was either

primarily associated with the teratoma or secondarily acquired by herniation through the interspace described by Ebner (cited by Watson[11]) between the coccyx and ischiococcygeus muscle.

Case 8. E. L., a 2-day-old baby girl, was admitted on April 17, 1942 because of an enormous cystic mass larger than the baby's head, which came from the sacrococcygeal area. This was attached by a very wide pedicle and over its surface ran large veins. The mass did not pulsate and no calcification could be seen by x-ray studies. The anal sphincter was intact. Because the mass was becoming necrotic, it was deemed advisable to remove it as a life-saving measure (Fig. 8A).

Operation. On April 18, 1942, under ether anesthesia, the enormous mass was

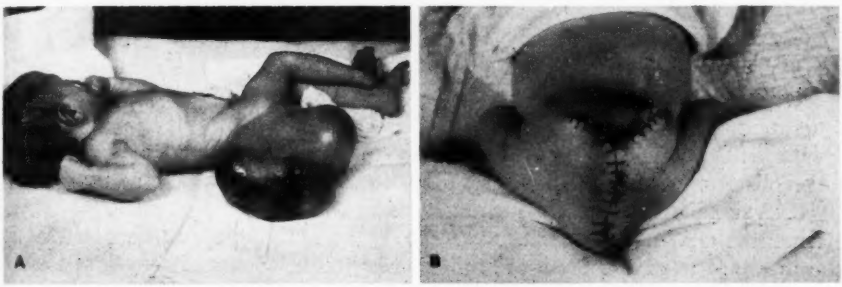

FIG. 8. *Case 8.* Pre- and postoperative photographs of ulcerating sacral teratoma.

circumscribed into healthy skin. The tumor was partially cystic with no evidence of deep infection. It was dissected from the sigmoid colon and rectum and was totally removed. It was seen to have arisen in the sacral region. In the sacrum there was a defect more on the right than on the left, the coccyx itself being absent. The incision below was carried to within $\frac{3}{4}$" of the anal sphincter. Some heavy subcutaneous tissue and some gluteal muscle were brought together over the rectum and sigmoid colon, giving firm support to these structures (Fig. 8B).

Postoperatively the child did remarkably well and is normal in all respects at the age of 7.

Pathological Examination. The removed specimen weighed $1\frac{1}{2}$ lbs. (baby's weight 8 lbs. before operation). Microscopically it was a trigerminal teratoma which contained cystic spaces lined by columnar epithelium, glial tissue, esophagus, bronchus with cartilage, thyroid, skeletal muscle and squamous epithelium.

Comment. The huge size that these lesions may assume is well shown in Fig. 7. Most observers in the past have believed that these sacral lesions were in all instances vestiges of a reduced parasitic twin. More recently, however, Patten[6] has pointed out another possible origin. The extraordinarily high incidence of trigerminal teratomata in precisely the region where the primitive streak of the embryo was formerly located may well mean that some of these masses originated by neoplastic growth of the tissue of this territory. In the embryo, the region of the primitive streak is a proliferation center from which cells entering all three of the germ layers are derived. It is quite logical, therefore, if it became involved in neoplastic growth that it would give rise to a teratomatous mass in which derivatives

of all three of the germ layers were represented. It should be emphasized that Patten's interpretation does not question the implication of twinning in a certain proportion of sacral teratomata. The possibility of an additional method of origin in this region, however, may well help explain the high incidence of teratomata in the sacral region as compared with other regions in which unequal conjoined twinning might be involved.

These lesions invariably are attached to the posterior surface of the rectum. The surgeon must consequently be warned against opening this structure. One should therefore keep oriented by having the assistant place a finger in the rectum of the infant during the operative procedure.

We have had 19 cases of similar sacral teratomas, only 3 of which were malignant.

SUMMARY

1. Large congenital defects of the scalp should be operated upon as an emergency procedure.

2. Mild surface infection in a myelomeningocele without sphincter paralysis or weakness of the extremities does not contraindicate surgery.

3. A congenital dermal sinus tract excised as a single specimen at operation shows continuity from the surface to the arachnoid and demonstrates the mechanism of the production of meningitis in such cases.

4. Diplomyelia or diastematomyelia is being recognized more frequently clinically. It becomes of surgical importance only when the cord is anchored by a midline bony mass preventing ascent of the cord from its fetal position.

5. Congenital defects of the neuroaxis in infants and children without gross neurological defects which appear hopeless at first are often amenable to operative procedures.

We are indebted to Dr. Bradley M. Patten for helpful advice and criticism in the preparation of this manuscript.

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TUMORS OF THE OCCIPITAL LOBE*

DWIGHT PARKINSON, M.D.,† WINCHELL McK. CRAIG, M.D., AND JAMES W. KERNOHAN, M.D.

Section on Neurologic Surgery and Section on Pathologic Anatomy, Mayo Clinic, Rochester, Minnesota

(Received for publication May 8, 1953)

FEW cases of tumors of the occipital lobe were recorded before the early part of the present century,[3,5,8,9,11,12,15,22,26,27,29,30,38] and no large series of cases of tumors confined exclusively to this lobe was reported before that of Horrax and Putnam in 1932.[24]

ANATOMY

Though often titled, divided and subdivided,[16,20,37] the occipital lobe is still without definite boundaries. In man this lobe is roughly pyramidal in shape having three dural surfaces: a lateral convex surface opposes the dura of the skull, a medial flat surface opposes the falx and an inferior sloping surface opposes the tentorium. Posteriorly, the three surfaces meet in a curved "point" corresponding to the cavity formed by the junction of the falx, tentorium and dura of the skull. It is generally agreed that the medial surface is bounded anteriorly by the parieto-occipital fissure. Books on anatomy differ as to the anterior limits of the other two surfaces. The lateral surface is bounded by an imaginary line drawn from the parieto-occipital fissure to the preoccipital notch, an indefinite landmark indicated by Ranson[31] as a fifth of the distance from the occipital to the temporal pole and by Gray[21] as a third of this distance.

The inferior surface is variously bounded. Gray used a perpendicular line from the preoccipital notch to the parieto-occipital fissure. Ranson depicted a perpendicular line from the tip of the calcarine fissure to the collateral fissure and from there on back, presumably to the level of the preoccipital notch.

Inasmuch as the calcarine fissure extends beyond its junction with the parieto-occipital fissure to a point just beneath the splenium and as the visual cortex has been definitely identified to this point, for the purpose of our study, Gray's arbitrary boundary line is slanted forward to extend from the preoccipital notch to the point where the calcarine fissure terminates beneath the splenium (Fig. 1).

In order to determine what proportion of the entire brain is constituted by the occipital lobe, a normal brain of an adult male body obtained at necropsy was fixed in formalin and sectioned. The cuts were made perpen-

* Abridgment of thesis submitted by Dr. Parkinson to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Neurosurgery.

† Fellow in Neurosurgery, Mayo Foundation, Rochester, Minnesota.

dicular to the cortical surface along the three boundaries as designated: Laterally, a line connecting the parieto-occipital fissure with the preoccipital notch which was taken as a point a third of the distance from the

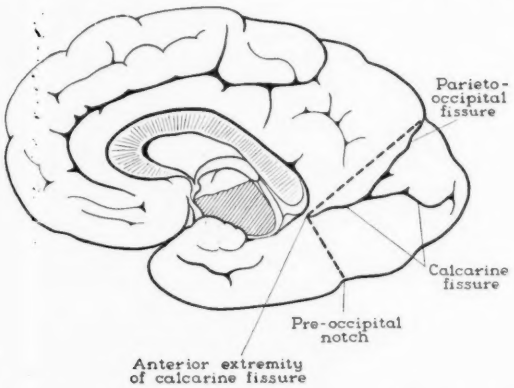

FIG. 1. Boundaries of the occipital lobe are indicated on the medial (falx) surface and on the inferior (tentorial) surface. The line for the medial surface extends straight from the dorsal edge of the parieto-occipital fissure to the anterior extremity of the calcarine fissure beneath the splenium. If the classic boundary formed by the parieto-occipital fissure and calcarine fissure were used on the medial surface, the striate cortex lying above the calcarine fissure along the anterior third would be excluded. The line for the inferior surface is seen slanting forward from the preoccipital notch to the anterior extremity of the calcarine fissure.

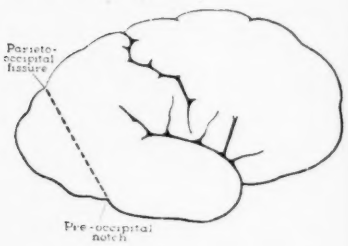

FIG. 2. The boundary on the lateral (skull) surface connects the dorsal end of the parieto-occipital fissure with the preoccipital notch, which is taken as a point a third of the distance from the occipital tip to the temporal tip. (This point is slightly too far forward in the drawing.)

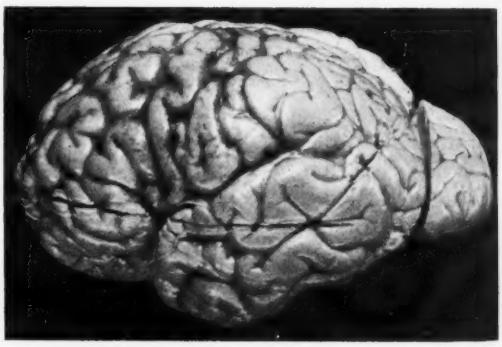

FIG. 3. Lateral view of normal brain demonstrating section connecting parieto-occipital fissure with preoccipital notch. Owing to obliquity, the point taken as the preoccipital notch appears less than a third of the distance to the temporal tip.

occipital to the temporal pole (Figs. 2 and 3); inferiorly, a line connecting the preoccipital notch with the anterior end of the calcarine fissure beneath the splenium (Fig. 1); and medially, a line connecting this point with the parieto-occipital fissure at its posterior boundary (Figs. 1 and 4).

When these cuts were completed the resected mass was found to be roughly a double pyramid with opposing triangular bases. The volume of the resected mass was 50 cc. and with its fellow occipital lobe it constituted one-thirteenth or 7.6 per cent of the volume of the supratentorial portion of the brain.

TYPES OF BRAIN TUMORS AND INCIDENCE

It is generally maintained that the occipital lobe is rarely disturbed by tumors. Unfortunately, for purposes of comparison, most of the cases reported either do not give exact locations or do not indicate whether pituitary body, pineal body, cerebellum or other portions of the brain are included.[6,13,33,35]

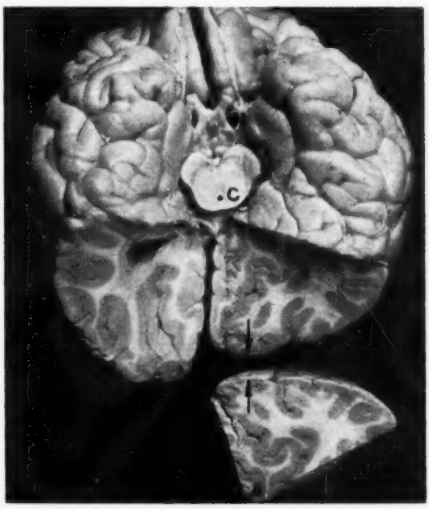

FIG. 4. Postero-inferior view of same brain as in Fig. 3. On the right the resected lobe has been tilted back as though hinged along the superior border (arrows). The apex of the triangle (A) fits into the point beneath the splenium, visible in the photograph just behind the collicular plate (C).

On the left a similar lobe was resected but sectioning was continued anteriorly along the calcar avis until all striate cortex was removed from deep in the curve of the lateral ventricle.

White,[36] in 1885, reported cases of 100 tumors of the brain none of which was limited to the occipital lobe, although it was involved in 6 of them. In 1899 Bramwell[7] found 2 of 40 tumors, or 5 per cent, to involve the occipital lobe. He included gummas. Clarke,[9] in 1916, reviewed 99 cases of intracranial tumors and found 4 in the occipital lobe: 3 gliomas and 1 meningioma. His series included pituitary tumors. The incidence of tumors in the occipital lobe would appear to be slightly more than 4 per cent.

Dowman and Smith[14] reported a series of cases in 1928 which could be broken down by definite location. Their series of 100 tumors included 10 pituitary, 2 pineal and 12 infratentorial tumors which left 76 supratentorial tumors with which the occipital lobe could compete for frequency of involvement. Of these, 2 tumors, or 2.6 per cent, were tumors of the occipital lobe.

In 1929 Frazier and Waggoner[19] reported the first sizable study of tumors limited to the occipital lobe, although 9 of their 40 tumors extended to the temporal lobe. It is impossible to state what percentage the 40 tumors represented since the total number was not given, but of the 40 tumors, 44 per cent were gliomas, 26 per cent fibroblastomas (probably meningiomas), 11 per cent cysts, 17 per cent tuberculomas and 2 per cent metastatic tumors.

One year later Allen[1] reported a series of 40 cases in which 24 tumors

were confined entirely to the occipital lobe. For the reason previously stated it is again impossible to state what percentage of a total this represents. Of Allen's 40 tumors 16 were gliomas, 4 endotheliomas, 3 sarcomas, 2 gummas and 1 was an angioma, and 1 a cyst. The remaining 13 were miscellaneous.

Elsberg[17] in 1931 reported on meningiomas only, and of a total of 102, listed 2 that were limited to the occipital lobe, being attached to the lateral sinus, and 13 more attached to the dura at the base which involved either occipital or temporal lobes.

In his book on intracranial tumors, published in 1932, Cushing[10] did not give the localization of all the tumors. He does break down the group to the extent that 366 infratentorial tumors, 360 pituitary tumors, 14 pinealomas and 92 craniopharyngiomas can be subtracted from his total of 2,023. This left 1,191 supratentorial tumors.

In the same year Horrax and Putnam[24] reported a series of 40 tumors of the occipital lobe from a total of 1,881 intracranial tumors. They discarded all tumors that extended beyond the occipital lobe, all multiple tumors and all tumors that involved both occipital lobes. Assuming that none of the 1,881 tumors were infratentorial, pituitary or pineal, 2.1 per cent of the series consisted of tumors of the occipital lobe.

It would seem justifiable to go further than this, however. The series of cases reported by Horrax and Putnam were part of the series reported by Cushing. Hence, of Cushing's 1,191 supratentorial tumors, excluding tumors of the pineal and pituitary bodies, at least 40 were confined to the occipital lobe. This amounts to 3.3 per cent in the largest series yet available.

The series of cases of Horrax and Putnam is the first in which tumors were confined entirely to the occipital lobe so that their study presents an incidence of this particular type of tumor which is of genuine value. Of the 40 tumors, 50 per cent were gliomas, 35 per cent were meningiomas and the remaining 15 per cent consisted of 2 angiomas, 1 hydatid cyst, 1 metastatic tumor and 2 unclassified tumors.

Elvidge, Penfield and Cone,[18] in 1937, reported on a series of 210 gliomas. Of these, 63 were subtentorial (including spinal and peripheral nerves) or pineal, which left only 147 to be considered as tumors located in the cerebrum. Unfortunately the group of 55 astrocytomas is the only one broken down beyond hemispheric distribution, and in this group 1 tumor was in the occipital lobe.

In a series of 223 intracranial gliomas, excluding infratentorial tumors and tumors affecting the optic nerve, Baker[2] listed 42, or more than 18 per cent, as occipital. Bennett,[4] in 1946, presented a detailed report of 446 primary intracranial tumors in a group of patients of military age. Of these, 75 were in locations other than the cerebrum, that is, in the pituitary or pineal body, infratentorial, and so forth. This left 371 tumors, of which 11 or 2.9 per cent were occipital. These 11 tumors included 2 meningiomas (of the total 39 meningiomas), 2 gliomas (1 of the 6 astroblastomas, 1 of the 13

spongioblastoma polares), 3 hemangiomas (of the total 30 hemangiomas) and 1 cyst (of the total 16 cysts).

Thus, from the few large series in the literature that could be analyzed it would seem that tumors of the occipital lobe constitute from 2 to 5 per cent of the supratentorial tumors eliminating tumors of the pituitary body, pineal body, and so forth. It must be remembered, however, that unless specified otherwise, designation of the lobe in which the tumor is located in

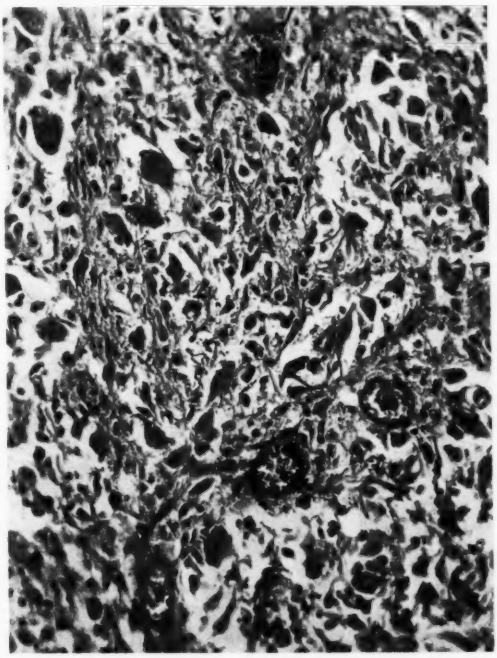

FIG. 5. Deep-seated astrocytoma, grade 2, in the right occipital lobe of a 59-year-old man. Considerable pleomorphism is exhibited but there is minimal if any vessel change and no mitoses are present (hematoxylin and eosin, ×235).

the series mentioned is probably on the basis of major rather than exclusive localization.

The type of tumor and its incidence, as obtainable from the few reports that lend themselves to such analysis, would average roughly as follows: gliomas, 45 per cent; meningiomas, 25 per cent, and a wide variety of tumors[23,25,32] none of which occurred in significant portion, 30 per cent.

At the Mayo Clinic in the past 25 years 275 cases of primary tumors involving the occipital lobe were reported. All metastatic and multiple tumors were excluded from this figure. These 275 cases were reviewed from the point of view of exact confinement to the occipital lobe in so far as surgical reports

and specimens removed at necropsy allowed. All cases in which extension occurred beyond the boundaries previously given (Figs. 1 to 4) were discarded.

There remained a group of 50 primary tumors strictly limited to the occipital lobe (inadequate tissue for diagnosis excluded 9). These 50 tumors formed the basis of our study. They comprised between 2 and 3 per cent of

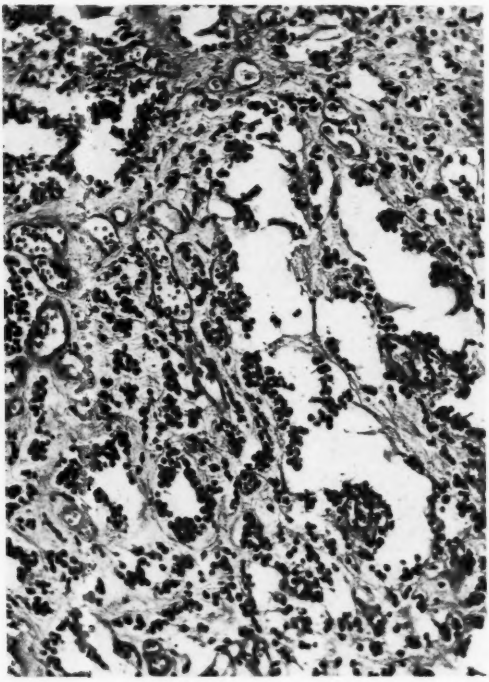

FIG. 6. Myxopapillary type of ependymoma, grade 1, on lateral surface of occipital lobe of 43-year-old man. There was strand-like calcification noted by roentgenogram 8 years previous to operation. The patient was alive 12 years postoperatively. Pleomorphism, anaplasia or mitoses were not present (hematoxylin and eosin, ×145).

the total supratentorial tumors over the same period after subtracting the pineal and pituitary groups.

Nineteen of the 50 tumors, or 38 per cent, were meningiomas, 6 of which were malignant. Thirteen, or 26 per cent, were glioblastomas; 2 were astrocytomas; 2 were astroblastomas, and 1 was an oligodendroblastoma. There were 6 ependymomas, making a total of 24, or 48 per cent, belonging to the glioma group.

According to the classification of Svien, Mabon, Kernohan and Adson,[34] 17 of these 24 gliomas were astrocytomas; 2 were astrocytomas grade 1, 2 grade 2 (astroblastomas) (Fig. 5), 11 grade 3 (glioblastoma multiforme) and

2 grade 4 (glioblastoma multiforme). Of the 6 ependymomas 1 was of grade 1 (Fig. 6), 1 of grade 2 (ependymoblastoma), 3 of grade 3 and 1 of grade 4.

The remaining 7 of the 50 tumors in the occipital lobe included unclassified gliomas, a fibrosarcoma (Figs. 7 and 8), a hemangiosarcoma and an osteogenic sarcoma.

The tumors were distributed evenly between the sexes within limits of expected error for such a small group, there being 23 males and 27 females. This relative proportion holds throughout the various types of tumors.

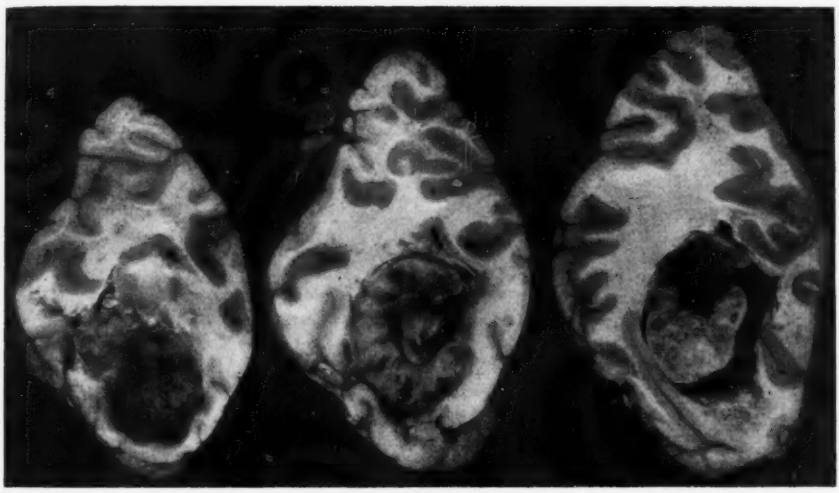

FIG. 7. Serial sections of occipital lobe of 20-year-old man show a small fibrosarcoma which did not reach the surface at any point.

Thus it can be seen from the literature and from our cases that primary tumors rarely involve the occipital lobe exclusively. The percentage of tumors affecting the occipital lobe only (2 to 3 per cent) is considerably less than the percentage of the supratentorial brain occupied by the occipital lobe (7.6 per cent by volume). Many tumors, however, are larger than the occipital lobe (50 cc.) by the time operation or necropsy is performed, and hence they would extend beyond this small, fairly silent lobe before data were secured in any reports for statistics.

The 275 cases in which the occipital lobe was involved by tumor represent a much higher percentage of the total brain tumors than the percentage of the supratentorial brain which is occupied by the occipital lobe. Thus, it is felt that the occipital lobe is probably involved by tumor as often as any other region of equal volume in the cerebrum. Because of its small size, however, it is rarely the exclusive site of a primary brain tumor.

The types of tumors found in the occipital lobe include practically all types of supratentorial tumors, excepting, of course, those that affect only

certain portions of the brain such as pineal and pituitary bodies. The vast majority of occipital tumors are gliomas and meningiomas.

CORRELATION

Correlation of the average total duration, that is, preoperative duration of symptoms plus postoperative survival, with the grade of each tumor gave the following results: (1) for astrocytomas of grade 1, 312 months, those of

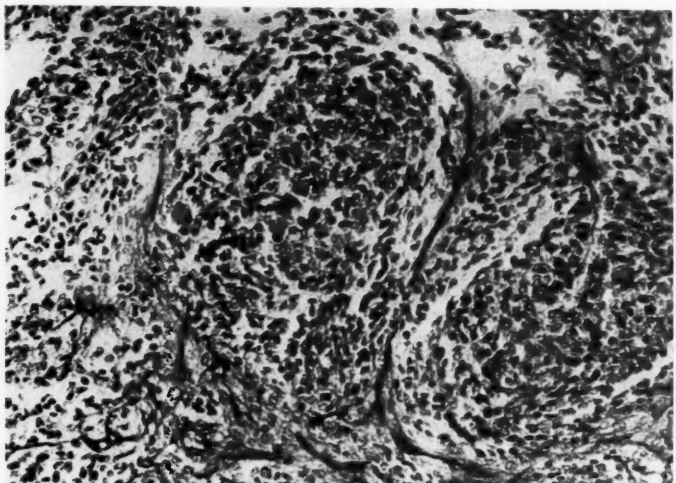

FIG. 8. Marked anaplasia and hyperchromatism of many nuclei of tumor shown in Fig. 7. A small amount of collagen is present (hematoxylin and eosin, ×185.)

grade 2, 57 months, those of grade 3, 18 months, and those of grade 4, 9 months; (2) for ependymomas of grade 1, the average history covered a total of 230 months, those of grade 2, 132 months, those of grade 3, 27 months, and those of grade 4, 2 months.

These two types of tumors thus demonstrate the same general pattern as those presented by Svien and associates[34] for the astrocytomas and by Mabon and associates[28] for the ependymomas although the duration for any one grade differs slightly. This is no doubt explainable by the fact that the number of tumors in each grade is too small in this series to yield a representative average.

The total duration from onset of symptoms plus postoperative survival for the group having benign meningiomas averaged 207 months and for malignant meningiomas, 46 months. It should be mentioned that in many instances the last entry on the record of a patient having a meningioma was to the effect that he was still living, and this date was used to figure the postoperative survival time. In the cases of malignant meningiomas and the

gliomas a higher proportion of the survival times represented actual time until death. Hence duration of the condition for the benign group errs on the low side.

COMMENT

An occipital lobe severed along the most definite boundaries established by the anatomists while making allowance to include all the visual receptive cortex, is about 50 cc. in volume and with its fellow represents about 7.6 per cent of the supratentorial portion of the brain.

Reports from the literature that contain information relative to the exact frequency of involvement of the occipital lobe by tumor are rare. Most of the large series of cases reported do not indicate how many tumors were occipital or what proportion of an entire series the enumerated occipital lobe tumors constitute. The series of cases reported by Horrax and Putnam was the only one of any size in which the tumors were confined entirely to the occipital lobe. Hence, it is the only series in which the incidence of this type of tumor is of genuine value.

From the few available figures in the literature, tumors of the occipital lobe appear to constitute between 2 and 5 per cent of the supratentorial tumors excluding pineal and pituitary tumors. There is a close agreement between the figures in our series and those of Horrax and Putnam for the two main groups of tumors, meningiomas and gliomas. In our series of cases 48 per cent of the tumors were gliomas; in Horrax and Putnam's series 50 per cent. In our group 38 per cent were meningiomas of which 6 or 12 per cent of the 50 tumors were malignant, and in Horrax and Putnam's series 35 per cent were meningiomas.

When the percentage of cases of brain tumor in which the occipital lobe only is involved by tumor (2 to 3 per cent) is compared with the percentage volume of the supratentorial portion of the brain occupied by the occipital lobe (7.6 per cent), it seems that the occipital lobe is rarely involved by tumor. If, however, the total number of cases in which the occipital lobe is involved is considered, the ratio is overbalanced the other way. It is concluded that the occipital lobe is probably involved by tumor as frequently as any other region of similar size (50 cc.) of the cerebrum. It is rarely exclusively involved because most tumors attain a greater size than may be contained in the occipital lobe and invade other tissue before they reach surgical attention. The peritorcular meningiomas are a notable exception. While it is true that they may attain a size of 200 cc. or more before surgical attention is necessary, they remain limited to the lobe as they frequently push the lobe ahead of them without involving any other region of the brain (Fig. 9).

SUMMARY

The occipital lobe comprises about 7 per cent of the supratentorial portion of the brain. Tumors limited exclusively to this lobe comprise about 3

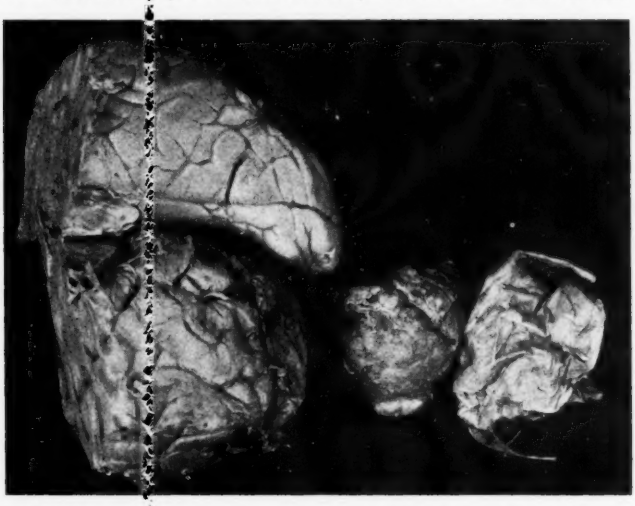

FIG. 9. Peritorcular meningioma, viewed from below, which involved the right occipital lobe of a 48-year-old man. Note the small area of dural attachment and the marked compression of the adjacent occipital pole.

per cent of the tumors of the supratentorial portion of the brain minus pineal and pituitary tumors.

About five times as many tumors involve the occipital lobe without being confined to this one lobe as those which remain within the lobe. This study indicates that the occipital lobe is involved by tumors as frequently as any other portion of the cerebrum of like size.

About 50 per cent of the tumors affecting the occipital lobe are gliomas, about 35 per cent are meningiomas and the remaining 15 per cent may be any of the tumors to which the cerebrum and supporting structures are subject.

The correlation between the degree of malignancy and the duration in our cases was comparable to that for other studies.

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THE APPLICATION OF NEUROANATOMICAL DATA TO THE DIAGNOSIS OF SELECTED NEUROSURGICAL AND NEUROLOGICAL CASES*

ELIZABETH CAROLINE CROSBY, PHD., SC.D.
Department of Anatomy, University of Michigan Medical School, Ann Arbor, Michigan

(Received for publication July 21, 1950)

IT IS a very great and deeply appreciated honor to be the first speaker on the Max Minor Peet Lectureship. Dr. Peet's international reputation as one of the great neurosurgeons of his day is a matter of common knowledge. His diagnostic skill, his wide experience, and his magnificent technique are most appreciated by those who worked with him, but everyone who knew him, not only in his own Department, at University Hospital and in the University of Michigan as a whole, but also in his professional and social contacts outside, felt the vivid personality and the lovableness of the man.

This lectureship was instituted by the group of neurosurgeons whom Dr. Peet had trained. They felt that it was the kind of expression of their devotion to him and of their unbounded respect for him that would give him great pleasure. Plans were made for a series of lectures dealing with various aspects of neurosurgery and these plans had been almost completed at the time of Dr. Peet's death.

The subject for the first lecture was chosen because anatomy was one of Dr. Peet's many scientific interests, and his practical knowledge of it, particularly in its gross aspects, was astonishing. His interest in anatomy extended far beyond the application of anatomical data to his own field, to a real enthusiasm for anatomical teaching and research. He was never too busy to counsel the man in difficulties with an anatomical problem; he lectured, on occasion, to the classes in applied anatomy; and he not only permitted but actively encouraged those in charge of neuroanatomy to make ward rounds with the neurosurgery staff—a course followed since by Dr. Kahn. As a most fortunate and very grateful recipient of this privilege, your speaker believes that probably the most important benefits that a clinical field can confer upon those who teach or do research in related preclinical subjects are the opportunity of seeing the application of the facts with which they deal to the solution of actual cases and the inestimable privilege of acquiring first-hand knowledge of the kind of information most likely to be of value to those under their tutelage.

ILLUSTRATIONS OF THE APPLICATION OF NEUROANATOMICAL DATA TO CLINICAL DIAGNOSIS

Consideration may now be given to the application of neuroanatomical

* The first lecture of the Max Minor Peet Lectureship, given at University Hospital, October 21, 1949.

data to a few selected clinical cases.† In order to give continuity to the discussion, easily recognized, related types of clinical disturbance—the interference with or the loss of conjugate deviation of the eyes in the horizontal plane (or, in one case, the significant retention of such conjugate deviation, associated with the loss of upward eye movements)—have been chosen for discussion.

Interference with Normal Conjugate Deviation of the Eyes in the Horizontal Plane Together with Other Clinical Signs of a Ponto-bulbar Lesion

Anatomical Considerations. Conjugate deviation of the eyes in the horizontal plane depends basically upon the simultaneous contraction of the lateral rectus of one eye and the medial rectus of the other. As is generally known, the lateral rectus is supplied by the abducens nerve arising from the abducens nucleus (Fig. 1). The medial rectus is innervated by crossed and uncrossed oculomotor fibers (Bernheimer[5]) from the median portion of the oculomotor nuclear complex which is termed the nucleus of Perlia (Fig. 2). Each abducens complex (Fig. 2) consists of larger neurons, constituting the abducens nucleus proper, from which arise the motor fibers of the abducens nerve, and small cells, forming the parabducens nucleus, which send fascicles through the homolateral medial longitudinal fasciculus (Spiegel and Sommer[43]) to the nucleus of Perlia.

Various observers (Spitzer,[44] see Fig. 3, page 469; Spiegel and Sommer;[43] and others) have considered that the impulses concerned with the conjugate deviation of the eyes toward one side are mediated by the medial longitudinal fasciculus of the same side. Such relations are indicated in Figs. 2 and 3. That there is still some question about the matter is evident, however, since in an account of 7 clinical cases showing unilateral internuclear ophthalmoplegia (with postmortem findings in 1 case) Cogan, Kubik, and Smith[9] reported that the medial longitudinal fasciculus was involved a little cephalad to the abducens nucleus on the side of the ophthalmoplegia. By means of the connections through the medial longitudinal fasciculus, coordinate contraction of the lateral rectus of one eye and the medial rectus of the other becomes possible, and thus conjugate deviation of the eyes in the horizontal plane toward the side of the contracting lateral rectus occurs. Destruction of the parabducens nucleus or of the medial longitudinal fasciculus (Spiegel

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† The case histories were obtained through the courtesy of the staffs in Neurosurgery, Neurology, and Neuropathology. The kindly suggestions and encouragement of Dr. Edgar Kahn and Dr. Robert Bassett of Neurosurgery and the anatomists, Professors Gerhardt von Bonin, Davenport Hooker, Tryphena Humphrey, and Russell T. Woodburne, during the preparation for this lecture were most valuable. The research work in process of publication by Crosby and Henderson, Henderson and Crosby, and Furstenberg and Cranmer, from which data were drawn for the interpretation of some of the clinical cases discussed in this lecture, was made possible by generous grants from the A. B. Brower and E. R. Arn Medical Research and Scholarship Fund and the Alphonso Morton Clover Research Fund, which are under the jurisdiction of the Dean of the Medical School. To the donors of these funds and to Mr. Vicente Colón of the Department of Biology, University of Puerto Rico, who drew the figures, sincere thanks are due.

and Sommer;[43] and others) results in a loss of such conjugate deviation.

Under normal conditions there is a balance between the tonus of the medial and the lateral rectus muscles. If only one abducens nerve is injured

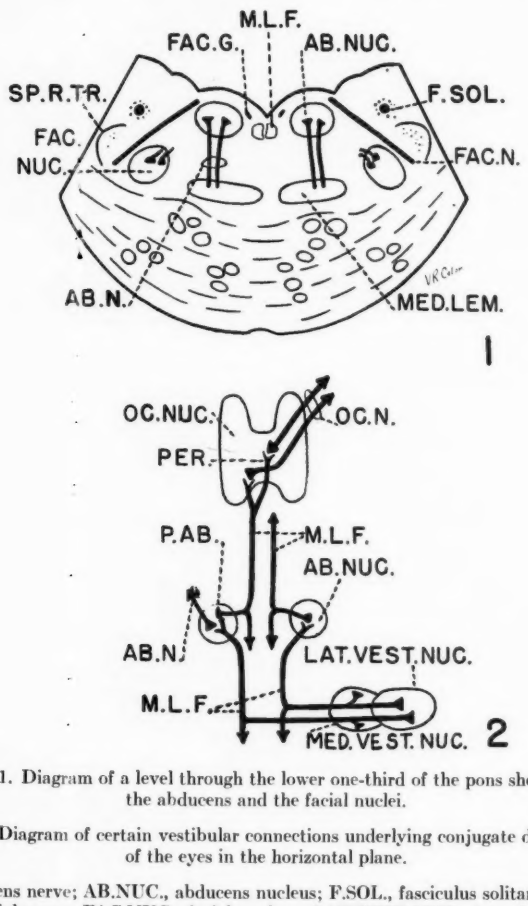


FIG. 1. Diagram of a level through the lower one-third of the pons showing the abducens and the facial nuclei.

FIG. 2. Diagram of certain vestibular connections underlying conjugate deviation of the eyes in the horizontal plane.

AB.N., abducens nerve; AB.NUC., abducens nucleus; F.SOL., fasciculus solitarius; FAC.G., facial genu; FAC.N., facial nerve; FAC.NUC., facial nucleus; LAT.VEST.NUC., lateral vestibular nucleus; M.L.F., medial longitudinal fasciculus; MED.LEM., medial lemniscus; MED.VEST.NUC., medial vestibular nucleus; OC.N., oculomotor nerve; OC.NUC. oculomotor nucleus; P.AB., parabducens nucleus; PER., nucleus of Perlia; SP.R.TR., spinal root of trigeminal nerve.

by the lesion, or if both abducens nerves are involved but not equally, there will be inequality in the tonus of the lateral recti of the two sides and diplopia will result. Moreover, when the fascicles forming the abducens root are partially destroyed, there is difficulty in turning the eyes toward the side of the lesion or in holding them in that position; and when the eyes are

so held on command, a nystagmus will occur (Spiegel and Sommer[43]). Ferraro and Barrera[14] found that, if the inner part (the so-called juxtarestiform portion) of the inferior cerebellar peduncle was destroyed within the brain stem (their intramedullary section), hypotonicity and nystagmus resulted. Since they did not get these effects from their extramedullary section, which was higher in the peduncle above the brain stem, it is possible, as Fulton and Dow[19] pointed out, that their results were due to an involvement of the vestibular centers. However, in practically all vertebrates, there are cerebello-motorius fibers (Fig. 3) which arise from the nucleus fastigius of the cerebellum and, usually after partial decussation, swing down through the juxtarestiform part of the inferior cerebellar peduncle to terminate in the abducens nucleus. Impulses passing over such cerebello-motorius fibers play a part in coordinating and strengthening conjugate deviation of the eyes in the horizontal plane. Destruction of the nucleus fastigius, or of the cerebello-motorius path, will produce a hypotonicity of the eye muscles and probably a nystagmus when the eyes are turned far lateralward.

The eyes change the direction of their gaze with even the slightest head movement, rotating toward the right as the head is turned toward the left (or toward the left when the head is rotated to the right), downward if the head is tilted upward, and upward if the head is bent forward. The semicircular canals, set in the three planes of movement of the head, are the peripheral end-organs in which are registered changes in head position. The peripheral terminations of those sensory neurons having their cells of origin in the vestibular ganglion pick up impulses from the stimulated hair cells of the crista ampullaris of each semicircular canal. The axones of such neurons distribute these impulses to the homolateral vestibular area and to the cerebellum. The vestibular area lies close to the floor of the fourth ventricle at upper medulla oblongata and lower pons levels (Fig. 3) and consists of four major nuclear groups—a medial, a lateral, a spinal or inferior, and a superior vestibular nucleus. Thus, impulses set up in the crista ampullaris of the right lateral semicircular canal are carried to the right lateral and, possibly, to the right medial vestibular nuclei. From these nuclei, other neurons relay the impulses through the contralateral medial longitudinal fasciculus (Fig. 2) to the abducens and parabducens nuclei, with a resulting initiation of a conjugate deviation of the eyes toward the left. An irritative lesion in the right lateral vestibular area may produce a nystagmus with the quick component toward the side of the irritation. This has experimental and clinical confirmation (Furstenberg and Cranmer[21]). If the lesion is destructive rather than irritative and the right lateral and right medial vestibular nuclei have less than normal function, an imbalance will exist between the right and the left vestibular centers, with the left lateral and left medial vestibular nuclei dominant. This imbalance will be evidenced by a nystagmus with the rapid component away from the side of the involvement or toward the left. If the right lateral and right medial vestibular nuclei are very greatly affected by a lesion, or these nuclei are destroyed, the strongly

dominant left vestibular centers will produce a deviation of the eyes toward the right—that is, toward the side of the injury.

Case 1. The patient was a 16-year-old white male, who, in the early stages of his illness, exhibited a blurring of vision and considerable generalized weakness of the body. Then his parents noticed that his face was drawn slightly toward the left side.

On admission to the hospital he complained of nausea, vomiting, and headache and had an incomplete flaccid paralysis of the face on the right and some difficulty

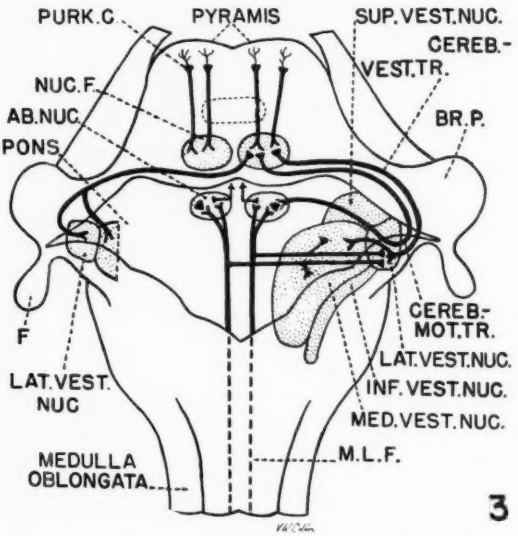


FIG. 3. Diagram of certain discharge fibers from nucleus fastigius of the cerebellum to the lateral and the medial vestibular nuclei and the abducens nucleus.

AB.NUC., abducens nucleus; BR.P., brachium pontis; CEREB.-MOT.TR., cerebello-motorius tract; CEREB.-VEST.TR., cerebello-vestibular tract; F., flocculus; INF.VEST.NUC., inferior vestibular nucleus; LAT.VEST.NUC., lateral vestibular nucleus; M.L.F., medial longitudinal fasciculus; MED.VEST.NUC., medial vestibular nucleus; NUC.F., nucleus fastigius; PURK.C., Purkinje cells; SUP.VEST.NUC., superior vestibular nucleus.

in swallowing, talking, and breathing. Taste was greatly diminished, everything tasting flat. His somewhat staggering gait was not made worse by closing his eyes and his sense of motion and position, vibratory sense, and tactile sensibility were normal on both sides. He had no abnormal movements or tremors and no incoordination in the finger-to-nose test. There was a considerable weakness of both abducens nerves, greater on the right, so that there was a marked diplopia, the patient seeing better with one eye closed. A nystagmus was evident on lateral deviation to both sides. There were no signs of increased intracranial pressure. The results of an otological examination were negative except that a slight deviation of the palate to the left was noted.

The patient gradually grew worse with increasing respiratory embarrassment.

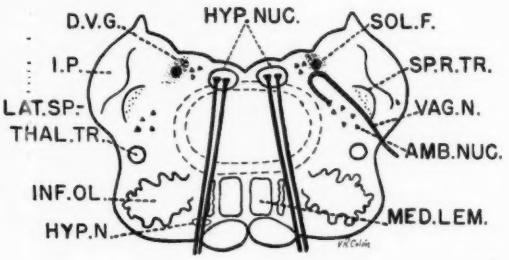
Ultimately he died a respiratory death. Postmortem examination revealed a tumor of the medulla oblongata and lower pons which was diagnosed by Dr. Scharenberg, with whose kind permission this case is reported, as a spongioblastoma unipolare or astroblastoma.

Anatomico-clinical Considerations. This case was obviously inoperable. It has been chosen for discussion here because it affords so excellent an illustration of the application of neuroanatomical data to the determination of the site, the gradual spread, and the final limits of a lesion—facts conceded to be necessary in all such cases for deciding whether operative procedure shall be carried out. The discussion of this case may well begin with a consideration of the underlying causes of the diplopia, since the foundations for such a consideration have been laid in the paragraphs preceding the case history. The abducens nuclei and the near-lying medial longitudinal fasciculi were spared by the lesion. Otherwise, there would have been a loss of conjugate deviation of the eyes in the horizontal plane. The weakness, or the decreased tonus, of the lateral rectus muscles was due to the partial destruction (greater on the right) of the abducens root fibers innervating them. This hypotonicity may have been accentuated somewhat by the destruction of the cerebello-motorius fibers as the inferior cerebellar peduncles were invaded by the tumor. The imbalance in tonus between the lateral rectus muscles of the two sides (the right side being more affected) was the underlying cause of the diplopia, which was indicated by the patient's ability to see better with one eye alone.

As is generally known, the root fibers of the facial nerve (Fig. 1), after their origin from the deeply situated facial nucleus at lower pontine levels, pass dorsomedialward to form a genu around the abducens nucleus and then turn ventrolateralward to emerge from the brain at the transition between pons and medulla oblongata. A destruction of the facial nucleus or its root fibers, or both, results in a flaccid paralysis of the face on the same side. The pathological material indicates that in this case the weakness of the muscles of facial expression on the right side was due to a partial involvement of both the right facial nucleus and the right facial root fibers. The hypotonicity of the muscles of facial expression on the right may have been increased somewhat by the destruction of cerebello-motorius fibers which discharge impulses from the cerebellum through the inferior cerebellar peduncle (here destroyed) to the facial nucleus.

Postmortem material indicates that the medial and the lateral vestibular nuclei (Fig. 3) were destroyed bilaterally. With their complete bilateral destruction there was no dominance of the vestibular area on either side. Consequently, the patient exhibited no conjugate deviation of the eyes to the right or left and no vestibular type of nystagmus. Nevertheless, the typical vestibular reflex of turning the eyes toward one side when the head is rotating in the opposite direction should have been, and probably was, interfered with by this lesion. Unfortunately, there is no record of its having been tested.

This patient showed a marked diminution of taste. In the normal individual impulses set up by gustatory stimuli are relayed to the brain from the anterior two-thirds of the tongue by sensory (special visceral afferent) fibers of the facial nerve, from the posterior one-third by similar components



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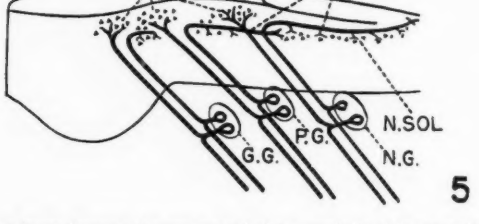

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FIG. 4. Diagram of a level through the upper half of the medulla oblongata. The approximate location of the scattered reticular cells related to inspiration and of those in the surrounding area related to expiration (see Pitts[38] and Magoun and Beaton[33] for the cat) is indicated by the concentric ovals of broken lines.

FIG. 5. Diagram illustrating the terminations of the root fibers of the facial, the glossopharyngeal and the vagus nerves which carry stimuli set up in taste buds and in viscera.

AMB.NUC., ambiguus nucleus; D.V.G., dorsal visceral gray; HYP.N., hypoglossal nerve; HYP. NUC., hypoglossal nucleus; G.G., geniculate ganglion; I.P., inferior cerebellar peduncle (corpus restiforme); INF.OL., inferior olivary nucleus; LAT.SP.-THAL.TR., lateral spino-thalamic tract; MED. LEM., medial lemniscus; N.G., nodose ganglion; N.SOL., nucleus solitarius or parasolitarius; P.G., petrosal ganglion; SOL.F., solitary fasciculus; SP.R.TR., spinal tract of trigeminal nerve; VAG.N., vagus nerve.

of the glossopharyngeal nerve, and from the epiglottis by like components of the vagal nerve. The cells of origin of such special visceral afferent fibers are in the ganglia (Fig. 5) on the respective nerve roots. The axons of the cells pass into the central nervous system to come into relation with a longitudinally running fiber tract, the fasciculus solitarius (Fig. 5), and to terminate in the so-called dorsal visceral gray. This dorsal visceral gray is a collection of small neurons situated near the levels of entrance of the

facial, the glossopharyngeal and the vagal nerves, along the ventricular side of the fasciculus solitarius. This dorsal visceral gray is the region of termination of fibers bearing impulses of gustatory significance and gives rise to secondary ascending fascicles which, after partial decussation, accompany the other components of the medial lemniscus to the dorsal thalamus. From the thalamus there is a relay to the cerebral cortex.

The remaining nuclear gray (nucleus solitarius or parasolitarius; Fig. 5) associated with the fasciculus solitarius receives impulses from various organs, notably the carotid sinus, and from the thoracic and some of the abdominal viscera. In the case under consideration there was a gradual bilateral destruction of the dorsal visceral gray at facial, glossopharyngeal, and vagal levels, and of the fasciculus solitarius and its associated nucleus solitarius (or parasolitarius). Taste was abolished with the destruction of the dorsal visceral gray, and respiratory changes and probably disturbances of other visceral functions indicated the inclusion of the fasciculus solitarius and nucleus solitarius within the lesion.

The patient's increasing difficulty in swallowing, speaking, and talking suggests a partial, progressive paralysis of the striated muscles of the tongue, the pharynx, the larynx, and the oesophagus. The hypoglossal nucleus (Fig. 4), which lies near the midline of the medulla oblongata beneath the floor of the fourth ventricle, and the hypoglossal roots, which extend ventralward from their nuclei to emerge on the ventral side of the brain stem and then distribute to the tongue muscles, were destroyed only in part by the spreading tumor. On either side of the medulla oblongata there was an increasing involvement of the nucleus ambiguus (Fig. 4) and the fibers arising from it which constitute the motor components of the vagal, the glossopharyngeal, and the bulbar accessory nerves innervating the striated muscle of the pharynx, the larynx, and the oesophagus. The paralysis of these muscles obviously interfered greatly with talking and swallowing. The speech difficulties were increased by the partial paralysis of the tongue. Since the choking became apparent just before the patient's entrance to the hospital and since, on entrance, the palate deviated to the left, it seems quite probable that, in the early stages, only the upper part of the right nucleus ambiguus was being invaded by the tumor. Then the tumor spread caudalward through the right nucleus and the left nucleus ambiguus was included also in the lesion. Sections made from postmortem material of this brain by Dr. Scharenberg show the bilateral involvement of these nuclei and of the root fibers arising from them but the destruction was not complete, since the patient did not need to be fed by tube but always retained some ability to swallow.

The ataxic gait was due primarily to the bilateral involvement of the inferior cerebellar peduncles (Ferraro and Barrera;[13] Fulton and Dow[19]). Neither the pathological material nor the clinical signs indicated the inclusion of the cerebellum itself in the affected area. In fact some of the clinical data argued against such an inclusion. Thus, there was no interference with

the finger-to-nose test, which is disturbed in lesions falling within the neocerebellum. Moreover, the patient showed no tremor. According to Bucy[8] (also, Grinker and Bucy[24]) interruption of the pathway from the dentate nucleus through the superior cerebellar peduncle to the contralateral ventral nucleus of the dorsal thalamus (from which impulses are relayed to motor and premotor areas of the cerebral cortex) will produce an intention tremor, as an accompaniment to voluntary movement. That this tremor may be due to imperfect compensation of other portions of the central nervous system, probably the cerebral cortex, has been pointed out by several observers (for example, Walshe[49]). Both ataxia and tremor were increased in a decerebellate dog in which a small lesion had been placed in the frontal region of the hemisphere (Luciani[32]) and in a monkey from which the part of the frontal lobe rostral to the motor area had been removed (Aring and Fulton[1]). However, removal of both cerebral hemispheres (and so the motor areas also) from a decerebellate cat has been shown to result in a disappearance of cerebellar tremor (Fulton, Liddell, and Rioch[20]). In the clinical case under discussion there was no known destruction either of the arc from the dentate nucleus to the cerebral cortex or of the frontal lobe and there was no tremor.

The acute respiratory embarrassment suffered by this patient was due to a variety of causes. In the first place there was an increasing paralysis of the larynx and the pharynx throughout the course of the disease. Moreover, there was bilateral involvement of the sensory root of the glossopharyngeal nerve and of the general visceral gray (nucleus solitarius or parasolitarius) associated with the fasciculus solitarius where these sensory root fibers end (Fig. 5). Consequently, there was an interference with impulses from the carotid sinus which exert an inhibitory effect on respiration. The pathways from higher centers (including those from the pontine respiratory center), which are related to the regulation of respiratory rhythm, were also involved in the lesion. The somewhat scattered reticular cells (Fig. 4), situated on either side of the midline dorsal to the inferior olivary nucleus throughout most of the medulla oblongata, and which are often said to form a respiratory center for maintenance of normal rhythm (Pitts,[38] Beaton and Magoun,[2] Magoun and Beaton[33]), also appear to have been destroyed to a considerable extent. There is a close relationship between the reticular elements serving inspiration and those functioning in expiration, one type being inhibited when the other type is functioning (Gesell[22]).

This tumor occupied the lower one-third of the pons and the greater part of the medulla oblongata, extending caudalward from a plane behind the level of entrance of the trigeminal roots to lower medullary levels. It had a limited extent in the pons, occupying the mid-portion of the area between the medial longitudinal fasciculi and the medial lemnisci (Fig. 1). Within the medulla oblongata the area included in the lesion increased. The tumor had approached nearer the ventricle and the hypoglossal nuclei had been included in part in the lesion (Fig. 4). Laterally it had spread

progressively, on either side, toward the outer surface of the medulla oblongata. At the time the neurological examination was made, the sparing of pain on both sides of the face and body indicates that the spinal roots of the trigeminal nerve and the lateral spino-thalamic tracts were relatively intact. However, the postmortem sections suggest that during the later course of the disease these fiber systems were becoming involved. The inferior cerebellar peduncles (Fig. 4) had been invaded by the tumor but the overlying cochlear centers were not included in the lesion, a fact in line with the results of the otological examination, which showed that the patient had no appreciable loss of hearing. Ventrally within the medulla oblongata the postmortem material shows a partial infiltration of the medial lemnisci and the pyramids (Fig. 4). At the time of admission to the hospital, however, the clinical examination did not indicate that the medial lemnisci were then involved, for tactile sensibility, vibratory sense, and motion and position sense were normal and closing his eyes did not affect the patient's staggering gait. No pyramidal tract signs were found on the earlier examination or later in the ward. In spite of the wide spread of this tumor, it was so intermingled with the remaining brain substance (small patches of which were undestroyed) that there was no outstanding evidence of increased intracranial pressure. Examination did not reveal choked discs.

Loss of Automatic Conjugate Deviation of the Eyes

Anatomical Considerations. Aside from eye movements initiated by the vestibular arcs, man has very few pure eye reflexes involving the extrinsic eye musculature. Nearly all eye movements are either voluntary or automatic, which implies that impulses reach the motor nuclei innervating eye muscles after a relay through the cerebral cortex (Crosby and Henderson[10]). Such a statement is in line with the fact that almost all visual impulses (in contrast to light reflexes and accommodation) are relayed by way of the visual thalamic center, the lateral geniculate nucleus, to the cerebral cortex and that only a very few optic fibers concerned with the more simple visual reflexes pass directly to the human superior colliculus. Certain cortical areas concerned with automatic eye movements lie in area 18 (Fig. 6), which adjoins the visual cortex in the occipital portion of the cerebral hemisphere, and in area 19 (Fig. 6) which borders it rostrally. Foerster[16,17,18] showed that stimulation of area 19 in man produces a conjugate deviation of the eyes toward the other side. It seems probable that such automatic deviation of the eyes, due to a discharge from area 19, is illustrated by a case described by Penfield and Erickson[36] (page 181). In this case the patient had a softening of the right posterior temporal region. During an epileptic seizure induced for the purpose of localizing the lesion, his eyes turned toward the left. Clinicians find that patients having a bilateral destruction of area 8 exhibit an inability to look voluntarily to either side but they can read by placing the index finger under a line of print and then following the finger as it moves slowly and steadily toward the other side of the page. The

eyes are then fixed on the end of the line unless the finger is moved back slowly to the beginning of the next line. In this fashion only can the reading be continued. This example illustrates the following movements and the fixation of the eyes which are the functions of area 19.

Experimental studies on monkeys (Crosby and Henderson[10]) demonstrated that there is a pattern for eye movements in area 19 impressed upon it by the terminations of its efferent fibers in relation with specific groups of neurons in the superior collicular region or the tegmental area of the midbrain. This pattern is illustrated in Fig. 6, which shows regions concerned with upward gaze (1, Fig. 6), with turning of the eyes upward and toward

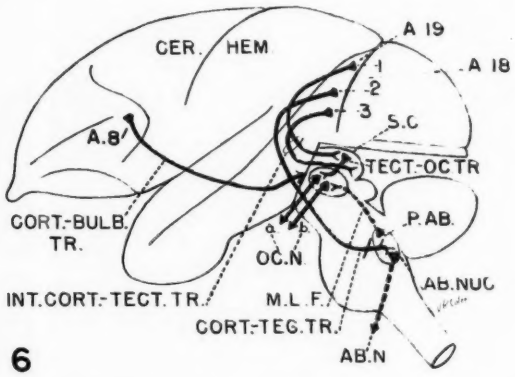

FIG. 6. Diagram illustrating certain connections of area 19 related to automatic eye movements in various planes.

1, 2, 3 are explained in detail in the text. A.8, area 8; A.18, area 18; A.19, area 19; AB.N., abducens nerve; AB.NUC., abducens nucleus; CER.HEM., cerebral hemisphere; CORT.-BULB.TR., cortico-bulbar tract; CORT.-TEG.TR., cortico-tegmental tract; INT.CORT.-TECT.TR., internal cortico-tectal tract; M.L.F., medial longitudinal fasciculus; OC.N., oculomotor nerve fibers (a) concerned with innervating the levator palpebrae superioris and the superior rectus and (b) with innervating the medial rectus; P.AB., parabducens nucleus; S.C., superior colliculus; TECT.-OC.TR., tecto-oculomotor tract.

the other side (not numbered), with conjugate deviation to the contralateral side in the horizontal plane (2, Fig. 6), then with deviation downward and toward the other side (not numbered), and finally with downward eye movement (3, Fig. 6). All of these deviations are of a conjugate type. Stimulation of the lower part of area 18 in the monkey results in eye movements upward and toward the other side (Walker and Weaver;[48] Crosby and Henderson[10]); stimulation of the upper part of area 18, in eye movements downward and toward the other side. Stimulation of the area intermediate between these two regions tends to give conjugate horizontal deviation. It will be noted that the upper part of area 19 and the lower portion of area 18 produce like movements, as do the upper part of area 18 and the lower portion of area 19. Moreover, these parts of areas 18 and 19 which are con-

cerned in like movements distribute to the same general region of the superior colliculus. The front end of this collicular area (Fig. 6) throughout phylogeny is concerned with impulses from the superior visual fields (which are responded to by raising the eyes), its caudal portion with impulses from inferior visual fields (which are reacted to by lowering the eyes). The distribution of the internal cortico-tectal tracts of Crosby and Henderson[10] or the cortico-collicular tracts of Peterson and Henneman[37] to the superior colliculi has been documented recently by the latter authors. In Fig. 6, for

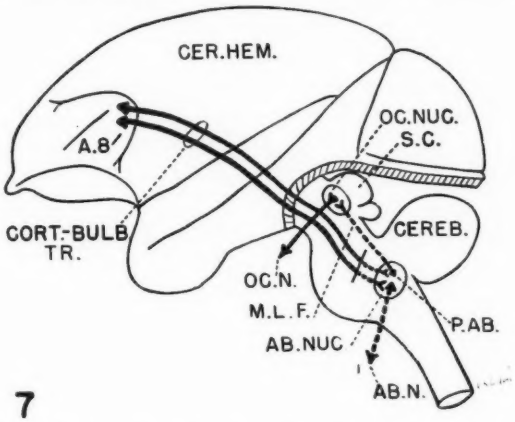

FIG. 7. Diagram illustrating the pathway for voluntary conjugate deviation of the eyes toward the other side.

A.8, area 8; AB.N., abducens nerve; AB.NUC., abducens nucleus; CER.HEM., cerebral hemisphere; CEREB., cerebellum; CORT.-BULB.TR; cortico-bulbar tract; M.L.F., medial longitudinal fasciculus; OC.N., oculomotor nerve; OC.NUC., oculomotor nucleus; P.AB., parabducens nucleus; S.C., superior colliculus.

purposes of simplicity, only the connections from area 19 are indicated. It can be seen that, through the internal cortico-tectal system, the upper part of each area 19 is connected with the frontal and medial portions of the superior colliculus and the lower part of area 19 is related with the caudal and lateral portions of this midbrain area. There is some crossing of these cortico-tectal fibers to the opposite side through the superior collicular commissure, which is not shown in the figure. No known fiber paths reach the superior colliculus from those portions of areas 18 and 19 concerned with horizontal deviation of the eyes. These portions discharge instead, as indicated for area 19 in Fig. 6, through a descending cortico-tegmental system which proceeds caudalward through the brainstem, probably with some synapse in course, to decussate and then terminate in the contralateral nucleus abducens (Fig. 6). Near its termination in nucleus abducens the cortico-tegmental tract (Fig. 6) lies near the cortico-bulbar fibers (Fig. 7) which provide a pathway for voluntary motor responses. The superior colliculus, then, is an important region of synapse in the neuron chain further-

ing automatic vertical gaze but is not a part of the arc subserving automatic horizontal deviation of the eyes. Cases of pineal tumor, or of tumor of the posterior part of the third ventricle, offer strong confirmation of this statement.

Case 2. This 15-year-old male was admitted to University Hospital as a patient of Dr. Peet. On examination he showed signs of increased intracranial pressure, some recurrent internal strabismus, indications of an early secondary optic atrophy, and a paralysis of automatic and voluntary upward gaze. An osteoplastic craniotomy was performed by Dr. Peet and an opening made into the third ventricle. Radiation was employed and for a time the patient showed some improvement, but, in spite of all efforts, succumbed. Postmortem examination by Dr. Wanstrom and Dr. Scharenberg confirmed the clinical diagnosis of pinealoma.

Anatomico-clinical Interrelations. The recurrent internal strabismus exhibited by this patient appears to have been due to pressure on the abducens roots, which, during their intracranial course after emerging from the brain, are liable to be involved when there is a great increase in intracranial pressure (Grinker and Bucy[24]). The paralysis of upward gaze was of both voluntary and automatic types. The automatic type was due to the pressure of the pinealoma upon the front part of the superior colliculi, with consequent interference with the terminal distribution of the internal cortico-tectal tracts to this region from the upper parts of areas 19 and the lower parts of areas 18. Voluntary upward gaze was lost when the pressure had increased sufficiently so that the cortico-bulbar fibers turning upward to enter the front end of the oculomotor nuclei (or the nuclei themselves in part) were also involved. In cases of pinealoma loss of voluntary upward gaze occurs later and, if the patient improves, usually disappears earlier than loss of automatic upward gaze. Complete permanent paralysis of upward gaze of reflex, automatic, and voluntary types will appear if the appropriate portions of the oculomotor nuclei are destroyed by the increased pressure, but such complete loss will be accompanied, at least in time, by signs of a flaccid paralysis of the superior levators of the lids and the superior rectus muscles.

If the patient had lived and the symptoms had not been relieved, there might have developed a loss of automatic downward gaze as the more caudal portions of the superior colliculi became involved, or increased pressure farther forward, compressing the pretectal nuclei (Kuhlenbeck and Miller[28]), might have led to a loss of light reflexes. He did not show any interference with automatic conjugate deviation of the eyes in the horizontal plane. This sparing of automatic horizontal deviation, in conjunction with the loss of automatic vertical movements, places the involvement at midbrain levels. Cortical lesions to produce such a case history must be bilateral, involving like specific portions of areas 18 and 19 but excluding the intervening areas associated with horizontal movements (Fig. 6)—unlikely lesions! At superior collicular levels the various systems dealing with automatic vertical movements of both eyes are brought together in the field and at the same time separated from those concerned with automatic horizontal movements.

Interference with Voluntary Conjugate Deviation of the Eyes in the Horizontal Plane Due to a Cortical Lesion

Anatomical Considerations. It has been recognized generally, due to the work of Ferrier,[15] Beevor and Horsley,[3] Levinsohn,[30] Brown,[7] C. and O. Vogt,[45] Smith,[42] and many others on the cortex of lower monkeys, of Beevor and Horsley[3] and Leyton and Sherrington[31] on that of the gorilla, of Grünbaum and Sherrington,[25] Leyton and Sherrington,[31] Bender and Fulton,[4] Dusser de Barenne, Garol, and McCulloch[11] on the cortical areas of the chimpanzee, and of C. and O. Vogt,[45,46] Foerster,[16,17,18] Penfield and Boldrey,[35] Rasmussen and Penfield,[40] Smith,[42] and others on the cerebral cortex of man, that voluntary eye movements in the horizontal plane (and sometimes in other planes), and often associated with a turning of the head, can be elicited from area 8 of the frontal lobe (Fig. 7). Some observers have noted also a dilatation of the pupils when certain portions of this cortical area were stimulated. Area 8 is fairly extensive; for the literature and some of the later accounts see Richter and Hines[41] and Walker[47] for the monkey, von Bonin[6] for various subprimates and primates including man, and von Economo and Koskinas[12] for man. It extends onto the medial surface of the hemisphere as well as onto its lateral surface in relation to area 6. There has not been general agreement as to its limits (compare the figures of C. and O. Vogt[45] or Foerster[16,17,18] with those of von Economo and Koskinas[12] and von Bonin[6] for the human brain). However, there is rather general agreement that the back end of the middle frontal gyrus, which is conceded to give horizontal eye movements on stimulation, falls at least in part within area 8.

In man, Foerster[16,17] obtained conjugate deviation of the eyes toward the opposite side when areas $8\alpha\beta\delta$ (the back part of the middle frontal gyrus) and 9 of C. and O. Vogt[45,46] were stimulated. He found that a stronger stimulus was needed to obtain the response from area 9 than from area $8\alpha\beta\delta$. Rasmussen and Penfield[40] not only produced eye movements from stimulation of the human area 8 but also such movements (in addition to closing the eyelid) from the portion of area 4 near the face area. Penfield and Erickson[36] reported case histories showing, among other clinical signs, interference with area 8 on one side during an epileptic seizure, so that the patient turned his eyes away from the side of the involvement. In lesions of area 8, such as those which sometimes occur in frontal lobe tumors or frontal leucotomies or from hemorrhage or skull fracture, the patient, on command, is unable to turn the eyes—can only turn the head—toward the side opposite the lesion. This condition is transitory, although it disappears more quickly in lower primates than in man. If area 8 is involved bilaterally, the eyes look straight ahead and the patient turns his head, but not his eyes, when asked to look to either side. Such a patient gives the usual responses to caloric tests, proving that the vestibular reflexes are normal. He can also follow a moving point from side to side if it is moved slowly and not jerkily so that fixation is retained (Holmes[27]). This is an indication that the cortical

areas concerned with automatic eye movements are intact although the voluntary eye movements are lost.

Observers (Beevor and Horsley,[3] Hirasawa and Kato,[26] Levin,[29] and others) have destroyed parts of area 8 and then determined the position and termination of its discharge fibers. So far as is known no worker has carried fibers to the abducens nuclei. In 1868 Prevost[39] pointed out that the hemiplegia resulting from a lesion of the internal capsule may be accompanied, during its early stage, by a deviation of the head and eyes toward the side of the lesion. Godlowski[23] (see also Levin[29]) traced such a path to the tegmental area of the midbrain but was unable to follow it beyond that level. He suggested that it synapses in course with secondary neurons which complete the arc to the abducens nucleus. In the monkey, Mettler[34] traced fibers to thalamic and midbrain centers and as far caudalward as nuclei pontis. Spiegel and Sommer[43] discussed the possibility that the fibers from area 8 terminate in the vestibular area and that this vestibular area then relays the impulses forward through the contralateral medial longitudinal fasciculus to the appropriate abducens nucleus.

In the laboratory at Michigan, area 8 was exposed in a macaque and, by stimulation, portions of it concerned with the horizontal deviation of the eyes were determined. These portions were then cauterized and the wound closed. After about 2 weeks the animal was sacrificed and the brain prepared for study by the Marchi technique. Fig. 7 shows the course of the bundle through the rostral part of the genu of the internal capsule and the base of the midbrain into the pontine tegmentum where, after partial decussation, the fibers reach a position ventral and ventrolateral to the abducens nucleus. Here they lie in relation with the cortico-tegmental fascicles from areas 18 and 19, both bundles distributing to the abducens complex. There is some evidence that a few fascicles of the cortico-bulbar system of the macaque do not cross the midline but terminate in the homolateral abducens nucleus. The bilateral projection of each area 8 on the abducens and parabducens nuclei would explain the relatively rapid recovery from the effects of a unilateral lesion of this cortical area. There is evidence, soon to be published, that the human abducens nuclei have very similar connections with each area 8.

Case 3. This patient was a 27-year-old white woman. Her trouble had previously been diagnosed as possible brain tumor and then as probably catatonic schizophrenia. Pneumonia developed and she was sent to University Hospital. Neurological examination showed that she was semicomatose with head and eyes turned to the right. There was some nuchal rigidity and apparently a left homonymous field defect. The tongue showed a slight deviation to the right and the right pupil was larger than the left. The deep reflexes were brisk in both upper extremities, possibly a little brisker on the left. No deep tendon reflexes could be demonstrated in the lower extremities except a right Achilles. There was a positive Hoffmann on the left but the plantar responses were normal. No papilledema was found but there was a slight engorgement of the veins of the right fundus. The spinal fluid was grossly bloody and xanthochromic after centrifuge.

Dr. DeJong, who most kindly gave permission for the use of this case, made a diagnosis of subarachnoid bleeding due to an aneurysm or a vascular tumor. Postmortem examination showed a ruptured surface aneurysm of the left middle cerebral artery due perhaps to a vascular anomaly.

Anatomico-clinical Interrelations. The absence of papilledema and the presence of a bloody spinal fluid suggested a vascular involvement. The resultant softening probably was responsible for the somewhat confusing clinical signs, some of which did not permit the establishment of the precise site of the lesion, although they suggested an involvement of the motor areas. The two symptoms of most definite localizing value in this case were the persistent turning of the head and eyes toward the right, which implied an encroachment on area 8 (or on areas 18 or 19) and the dilatation of the pupil on the right which, in conjunction with the motor signs, is suggestive of an irritative lesion in the left area 8 (Fig. 7). It is true that dilatation of the pupil may be the result of an irritative or of a destructive lesion at several levels of the nervous system. Only when other clinical signs appearing in conjunction with it are taken into account does the dilatation have definite localizing value. That it may be of importance in this case is documented by the experimental work of Wilbur Smith[42] and others (see also the case report of Penfield and Erickson,[36] page 176). Smith found that, not only did horizontal conjugate deviation of the eyes toward the other side result from a stimulation of area 8 in the monkey, but that the pupils dilated during such a deviation. Under certain experimental conditions in the monkey (Crosby and Henderson, to be published) and probably under certain clinical conditions in man, one pupil may respond more than the other. This reaction of the right pupil indicates that the lesion is irritative and that it is on the left. An irritative lesion in the left area 8 produces deviation of the eyes and head toward the right. The vessel that supplies the left area 8 is the left middle cerebral artery. The facts suggest that this vessel is the site of the lesion, a localization documented by the postmortem findings.

CONCLUSION

The cases which have been discussed serve merely to illustrate a few possible applications of anatomical data to the solution of clinical problems. That the great wealth of available anatomical knowledge is not always at the disposal of the clinician is due partly to his lack of appreciation of its practical value, but largely to the failure of those of us who are working in neuroanatomy and teaching in medical schools to understand the diagnostic significance of the anatomical facts with which we are dealing.

Dr. Peet was an outstanding example of a neurosurgeon whose experience was great enough and whose vision was broad enough to enable him to draw from related fields those facts which enriched his knowledge and aided in the advancement of his profession.

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CASE REPORTS AND TECHNICAL NOTES

INTRADURAL LIPOMA OF THE SPINAL CORD

REPORT OF A CASE

TOKUSO TANIGUCHI, M.D., AND JOSEPH A. MUFSON, M.D.
St. Joseph's Hospital, and Marquette University School of Medicine, Milwaukee, Wisconsin

(Received for publication February 17, 1950)

In an excellent review of the literature, Ehni and Love[1] in 1945 collected 26 cases of intradural lipoma of the spinal cord and added 4 of their own. Since then 2 additional cases have been reported.[3,4] Intradural lipomas, unassociated with spina bifida, are very rare, constituting about 1 per cent of all tumors of the spinal cord.

In view of its infrequent occurrence, the following case of intradural lipoma of the spinal cord is being presented. The tumor was presumably intramedullary in location.

CASE REPORT

M. S., a 52-year-old white male, was admitted on July 25, 1949 with the chief complaint of progressive weakness in both lower extremities of 5 years' duration. For about 4 years prior to the onset of this difficulty, he had noted periodic twitchings of the muscles in the legs and spasmodic flexion at the hip and knee joints. Gradually, these flexion movements became forceful, especially on the left, causing involuntary flexor spasms of the lower limbs. He also noted progressive loss of sensation in the thighs. On July 15, 1949, he became totally unable to walk and subsequently lost sphincter control of the urinary bladder and bowel. He was thought to have syringomyelia by several physicians who had examined him since the onset of his symptoms.

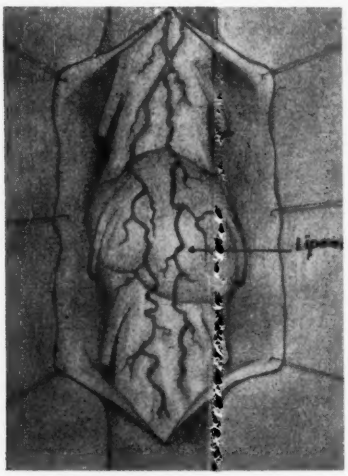

FIG. 1. Operative sketch showing the tumor mass in its relationship to the spinal cord.

Examination. He was a well developed, well nourished, middle-aged male who appeared in no acute distress. B.P. 208/140. The chest was clear and the heart sounds were regular. There was marked weakness of both lower limbs with atrophy of the quadriceps muscles. Fibrillations were noted in the atrophic muscles. Knee and ankle reflexes were hyperactive, and a Babinski sign was present bilaterally. There were hypesthesia and hypalgesia over the anterior and lateral aspects of the thighs corresponding to the 2nd and 3rd lumbar dermatomes. Vibratory sensation was absent in both lower limbs. Position sense was impaired in the toes. The abdominal reflexes were present in all quadrants. There were no neurological disturbances in the upper limbs, and the cranial nerve functions were intact.

Lumbar puncture on July 27, 1949 revealed a partial subarachnoid block. The CSF was crystal clear. There was a slight increase in the total protein (53.2 mg./100 cc.) with a normal amount of globulin. Wassermann and colloidal gold tests were negative.

Plain roentgenograms of the dorsal and lumbar spine were negative, except for moderate hypertrophic changes in the lower lumbar vertebrae. On July 28, 1949 pantopaque myelography disclosed an obstruction to the column of oil at the level of the 11th dorsal intervertebral space, suggesting the presence of an intraspinal mass.

Two days after myelography, an acute coronary thrombosis developed, and it was necessary to postpone surgery for several weeks.

Operation. On Sept. 29, 1949 a laminectomy of the 10th, 11th, and 12th thoracic vertebrae was carried out. Upon opening the dura, a fusiform swelling of the spinal cord was found at the level of the 11th thoracic vertebra (Fig. 1). This swelling was produced by a well circumscribed, lobulated, intramedullary tumor, measuring approximately 3×2 cm., which had

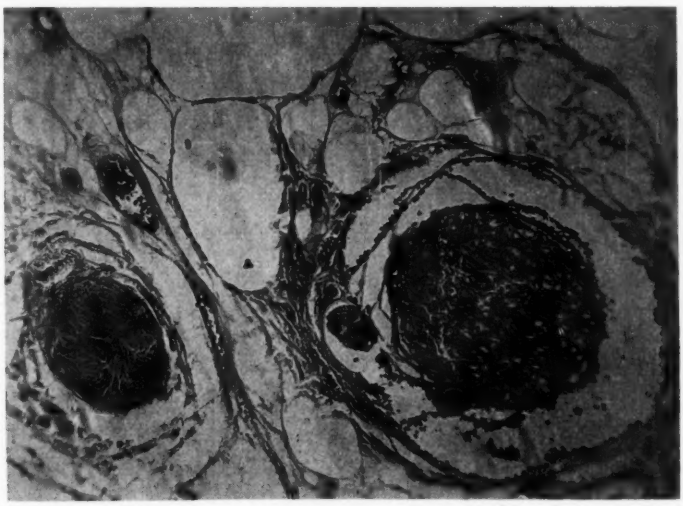

FIG. 2. Photomicrograph showing the tumor to be composed of well differentiated adipose tissue. Nerve bundles are seen traversing the fat tissue.

a distinct yellow color. The cord in this area was almost entirely replaced by the tumor, with only a small amount of neural tissue present anteriorly and laterally. The pia-arachnoid covered the posterior surface of the tumor. While the tumor appeared to be well circumscribed it was intimately associated with the surrounding neural tissue and emerging nerve roots so that no distinct line of separation could be made out. A small portion of the tumor was removed through a midline dorsal incision, but the major portion was left behind, since it was felt that the tumor could not be completely removed without sacrificing the cord at that level. The wound was closed, but the dura was left open for added decompression.

Histological Study. The tumor was composed of well differentiated adipose tissue. Nerve bundles could be seen traversing the fat tissue (Fig. 2).

The *postoperative course* was uneventful and he was discharged from the hospital on Oct. 15, 1949 without any change in the neurological status.

DISCUSSION

Intradural lipomas form a very interesting group of spinal cord tumors. They have a tendency to occur at three distinct age periods[1]—during infancy, at puberty and during the 3rd to 5th decades. The lipomas occurring during infancy are often

associated with multiple congenital deformities and may extend along the entire length of the cord. The distribution of the lipomas between sexes is about equal. They tend to arise predominantly in the lower cervical and upper thoracic regions and characteristically appear on the dorsal aspect of the cord with a predilection for the midline.

A review of the literature reveals only a few cases reported as intramedullary lipoma of the spinal cord. The true intramedullary nature of these lipomas has been questioned.[1] There is some basis for this skepticism, for although occasional fat cells have been described as being found within the leptomeninges by Chiari, Kollikes, Bostroem, and others, fat has never been found lying free within the neural tissue. In our case, although the tumor was intramedullary in location, no definitive statement can be made as to its site of origin due to the lack of histological sections showing the exact relationship between the tumor and the surrounding leptomeninges and neural elements.

In general, the intradural lipomas are composed of well-differentiated adipose tissue, similar to that found elsewhere in the body. Excess fibrous tissue may be present in some of the lipomas (fibrolipomas). A myolipoma containing many striated muscle fibers has been reported by Gowers.[2]

In regard to the histogenesis of lipomas of the spinal cord, several theories have been advanced: (1) metaplasia of adipose tissue from the connective tissue of the pia-arachnoid membrane; (2) developmental error in which lipomas arise from inclusions of embryonic rests within the meninges during the formation of the neural tube; and (3) proliferation of fat cells which are found occasionally in the pia. None of these theories seems to explain satisfactorily all the features of intradural lipomas. However, the weight of evidence seems to point to a developmental error, in view of the frequency with which lipomas are associated with spina bifida and other congenital anomalies. To account for the myolipoma and fibrolipomas, the possibility of these tumors being hamartomas must be considered.

In contrast to lipomas found in other parts of the body, those present intradurally in close relationship to the spinal cord are often removed with great difficulty or are only partially removable because of their intimate association with the neural tissue and nerve roots. In these cases, the symptoms are usually of long duration and may extend over a period of 10 years or more.

CONCLUSION

A case of intradural lipoma of the spinal cord having intramedullary features is presented. Up to this date, 32 previous cases of intradural lipomas have been described in the literature. "Intramedullary" lipomas are unusually rare, and their true site of origin is still a matter of dispute.

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LUMBAR EXTRADURAL HEMATOMA

REPORT OF CASE SIMULATING PROTRUDED DISK SYNDROME

HENDRIK J. SVIEN, M.D., ALFRED W. ADSON, M.D., AND HENRY W. DODGE, JR., M.D.*
Section on Neurologic Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

(Received for publication March 14, 1950)

The problem of low back pain and sciatica confronts the busy physician often enough so that most doctors, and, in particular, general, orthopedic and neurological surgeons, have formulated in their own minds a list of conditions to be considered when presented with this particular symptom complex.

Such a list usually includes protruded intervertebral disk in the lumbar region, neoplasms of the vertebrae, primary and metastatic neoplasms of the spinal cord, vascular anomalies of the cauda equina, prostatic, rectal or other pelvic neoplasms, spinal injury, spinal anomaly, spondylolisthesis, sciatic injury, infection or epidural abscess, tumor of peripheral nerve, arthritis, neuritis and acute back strain.

The wealth of conditions which the examining physician is able to review rapidly in his mind as he evaluates the diagnostic possibilities of a particular case is augmented by his clinical experience, and frequently by his reading. For this reason it is felt that the recording of this case of lumbar extradural hematoma following trauma in which the symptoms simulated those of a protruded intervertebral disk would be of interest.

The pertinent literature was reviewed by Ver Brugghen[2] in 1946. He was able to find only 8 cases of extradural spinal hemorrhage in addition to the 1 that he reported. Chavany and co-workers'[1] case of verified lumbar epidural hemorrhage in a woman 70 years old, and our own case add 2 more cases to the small number of cases of extradural spinal hemorrhage reported in the available literature.

REPORT OF CASE

The patient, a white man 67 years old, registered at the Mayo Clinic Oct. 7, 1948. His chief complaint was pain in the back extending down both legs. His family history was noncontributory. He had always been in good health until he slipped and fell on the ice in December, 1947. He landed in a sitting position on his buttocks, and felt no immediate pain or disability.

Two weeks later pain developed in both legs; it began in the thighs and extended into the calves. The pain was worse on the right than on the left side. He also experienced backache but this was not of a severe nature.

He described the pain as steady, usually dull but occasionally sharp. It was most noticeable on arising in the morning. After he was up and around for a while, the pain would be less severe. Standing and walking during the day were equally effective in bringing on the pain. Sitting would give some relief. Cough and strain aggravated the pain. No pain was noticed at night, and he had not experienced numbness, tingling or sphincteral difficulties.

Examination. B.P. was 120 mm. of mercury systolic and 85 diastolic. Pulse rate was 70, and respiratory rate 98. Periodontal sepsis, grade 2, dilated inguinal rings on both sides and prostatic enlargement, grade 1, were noted.

Weakness of the left anterior tibial and posterior tibial muscles was graded −2 on a grading basis of −1 to −4 (−1 represents least weakness); that of the left quadriceps muscle was graded −1. Decrease of the right quadriceps reflex was graded −1, of the left quadriceps

* Fellow in Neurosurgery, Mayo Foundation, Rochester, Minnesota.

reflex −2. Ankle jerks were absent bilaterally and the cremasteric and anal reflexes were decreased to grade −4. Babinski signs could not be elicited.

Sensory findings were normal. Sphincters, station and gait were normal. The Lasèque sign was positive on the right.

Laboratory tests on the blood revealed 12.7 gm. of Hb./100 cc.; 4,600 leukocytes/c.mm., sedimentation rate of 30 mm. in 1 hr. (Westergren). Urine was normal. Roentgenograms of the thorax gave negative results. Roentgenograms of the lumbar region revealed partial sacralization of the 5th lumbar vertebra on the right, residual opaque oil in the caudal canal, small areas of increased density over the acetabulum and osteo-arthritic changes in the right hip. Myelograms with radiopaque oil, made elsewhere a few days prior to the patient's registration at the clinic, demonstrated complete block to the cephalad flow of the oil opposite the upper border of the 3rd lumbar vertebra.

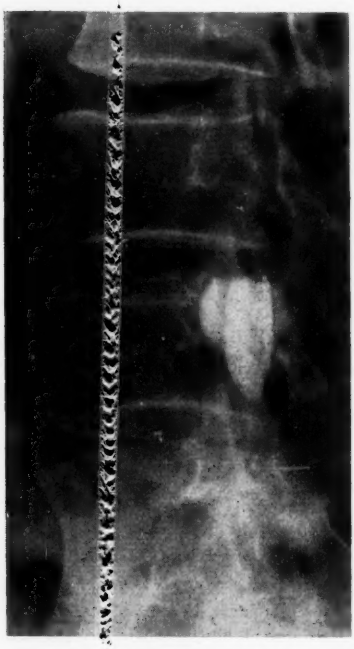

FIG. 1. Myelography with aid of radiopaque oil revealed complete subarachnoid block to cephalad flow opposite the upper border of the 3rd lumbar vertebra. Defect suggestive of an extradural lesion.

In view of the fact that the patient had sufficient residual radiopaque oil remaining from his earlier myelography to permit satisfactory roentgenoscopic examination, this procedure was carried out. This confirmed the block as previously described and suggested that the lesion producing the block was extradural (Fig. 1).

A diagnosis of protruded intervertebral disk at the 3rd lumbar interspace or spinal cord tumor was made and operation was advised.

Operation. Hemilaminectomy was carried out at level of 3rd and 4th lumbar vertebrae. As dissection was carried down to, and including, the lamina of the 3rd and part of the 4th lumbar vertebrae, a hard fibrous mass situated to the left and slightly dorsal to the dura was encountered. It measured 2 by 2.5 by 1 cm. This mass compressed the dural sac. As it was being separated from the dura, it was opened inadvertently and black substance, which appeared to be old blood, exuded. The mass, which was encapsulated, was completely removed and was diagnosed by the pathologist as being an old organized hematoma. The dura was opened and the cauda equina inspected; no abnormalities were noted. The CSF was clear.

Course. The patient had an uneventful convalescence. He was dismissed from the hospital on the 8th postoperative day. He was free of pain and has remained so.

SUMMARY

In a case of low back pain and sciatica following trauma the presented symptoms simulated those of intervertebral disk. Operation revealed the symptoms to be caused by lumbar extradural hematoma. Removal of the mass effected complete relief of symptoms.

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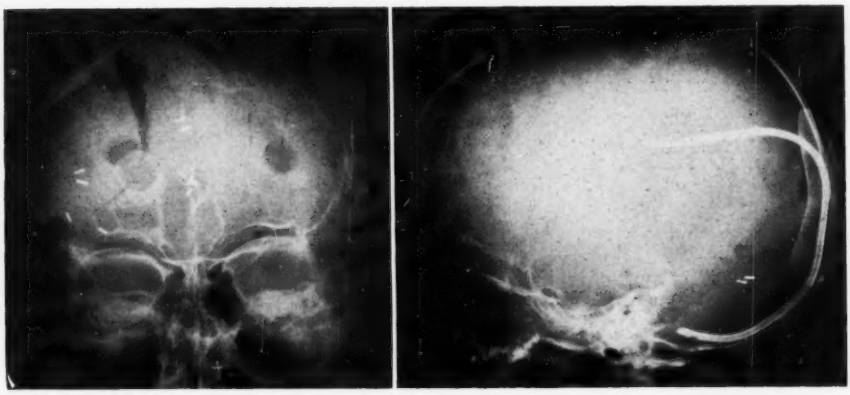
STAINLESS STEEL SCREEN FOR COVERING TREPHINE AND OTHER SMALL CRANIAL DEFECTS

JOSEPH RANSOHOFF, M.D.

Department of Neurological Surgery, College of Physicians and Surgeons, Columbia University, and the Neurological Institute, New York City

(Received for publication March 7, 1950)

The disfiguring depressions that occur over anterior burr holes, while certainly a trivial matter, can become a source of embarrassment and irritation to patients. In 1948 Mount[1] reported the technique of covering these defects with tantalum discs, and in the hands of many this method has been very successful. These discs, however, must be fitted carefully to the burr hole and have occasionally slipped



FIGS. 1 and 2. Stainless steel screen covering (*left*) anterior burr hole; and (*right*) occipital defect.

out of place, especially if the flap moves in its bed for one reason or another. In addition, tantalum discs are moderately expensive.

Early in 1948 the use of a screen of inert wire was suggested for the same purpose. I have attempted to trace the source of this suggestion and am unable to do so. The idea was not original and is merely being reported, as it has proven to be an extremely simple, inexpensive and effective method for handling this minor problem. It has been utilized in over 200 instances during the past year at the Neurological Institute and has also been employed by Davidoff and others in and about the New York area.

The stainless steel screen (size 0.020-18.8, Type 302), comes in large sheets which can be sterilized repeatedly without ill effect. After the bone flap is in place, a small square of screen is cut with heavy scissors just large enough to cover the defect to be hidden. This square is held in place over the burr hole by 2 or 3 periosteal sutures. The screen can be easily molded to the shape of the underlying skull by pressure with the use of gloved fingers. We have not experienced any difficulty with these screens, and they serve as a lattice for connective-tissue fibers which rapidly infiltrate the mesh, firmly binding together the screen, bone and periosteum. This sheet of connective tissue does not, however, grow down into the burr hole to

involve the underlying dura or brain, as we have learned when we have had occasion to re-operate upon patients in whom this screen has been utilized. Fig. 1 shows an x-ray of such a piece of screen covering an anterior burr hole.

We have also used somewhat larger segments of screen* to cover other types of small cranial defects with satisfactory results. Fig. 2 shows a fairly large occipital defect covered with screen which was held in place by suturing the periosteum over its edges. The defect was produced during the process of splitting the tentorium prior to the insertion of a Torkildsen tube in a hydrocephalic infant with an aqueduct stenosis and a subarachnoid block at the tentorial ring.

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* The screen can be obtained from Uza Nudell, 125 West 45th Street, New York City.

ENCEPHALOGRAPHY IN THE PRESENCE OF A TANTALUM IMPLANT

W. JAMES GARDNER, M.D.

Cleveland Clinic and the Frank E. Bunts Educational Institute, Cleveland, Ohio

(Received for publication March 6, 1950)

Tantalum appears to be the most satisfactory material for the repair of cranial defects, but one objection frequently raised to its use is that its radio-opacity interferes with subsequent encephalography. The degree of radio-opacity of tantalum, of course, is dependent upon the thickness of the plate employed. Many surgeons seldom use anything less than .015 of an inch in thickness. We have found that a sheet of tantalum .007 of an inch in thickness is perfectly adequate to repair a cranial defect of any size, and it has the following advantages. It can be more

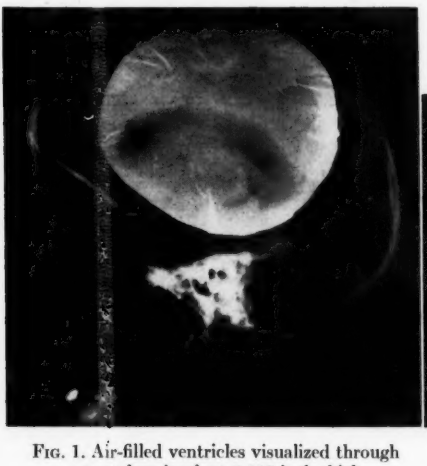

FIG. 1. Air-filled ventricles visualized through a tantalum implant 0.007 inch thick.

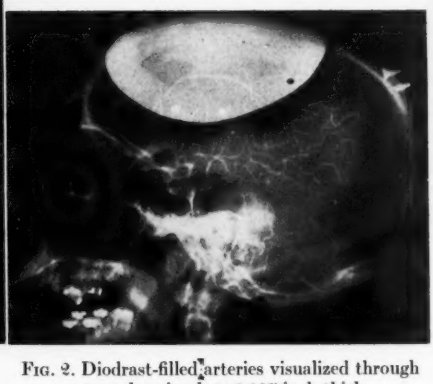

FIG. 2. Diodrast-filled arteries visualized through a tantalum implant 0.007 inch thick.

readily shaped at the operating table. It can be cut with a pair of scissors, and holes can be punched in it with a modified card punch. When properly shaped and fastened to the edges of the skull by means of tantalum screws, it affords perfectly adequate protection to the underlying brain. In addition to being more readily formed, it is less expensive than the thicker sheets, and by using more penetrating x-rays it is possible to obtain satisfactory visualization of the air-filled ventricles beneath the implant or of diodrast-filled vessels as shown in Figs. 1 and 2.

A NEW PERCUTANEOUS NEEDLE FOR ARTERIOGRAPHY

MALCOLM B. PARHAD, M.D.,* AND JAMES L. POPPEN, M.D.
Department of Neurosurgery, The Lahey Clinic, Boston, Massachusetts

(Received for publication April 10, 1950)

Because of certain difficulties which arose when the conventional straight 18 gauge intravenous needle was used for the closed method of arteriography, it was

FIG. 1. This needle has been used in over 200 cases in which carotid arteriograms were made. We have found it a great improvement over the straight needles. The needle is made of a strong and malleable material so that it can be adjusted to any angle. The tip is short and sharp; the proximal end is attached to a Luer-Lok syringe (Fig. 2).

FIG. 2. Same as shown in Fig. 1, with 10 cc. syringe and two-way petcock attached.

FIG. 3 (*Left*). The angle is shown at which the straight needle enters the artery. (From Poppen, J. L.: Aid of arteriograms in diagnosis and treatment of intracranial aneurysms. *Radiology*, 1949, *52:* 347–352, Fig. 2.)

FIG. 4 (*Right*). The angulated needle is shown in position lying in a plane horizontal to the artery rather than at the acute angle shown in Fig. 3. In this position, the needle is less likely to be displaced from the lumen of the artery by slight movement at the time of injection.

* Formerly Fellow in Neurosurgery. Now at American University of Beirut, Department of Surgery, Beirut, Lebanon.

deemed advisable to make certain modifications of the needle. The ideas of Simonson were incorporated. Plans were drawn for a needle that would adjust itself to the variations in the anatomical structures of the necks of patients to be subjected to arteriography. The underlying principles in executing a satisfactory arteriogram make it important that the distal opening of the needle lies parallel to the artery that is to be injected. It is advisable that the needle be adjusted to the desired angle according to the needs in a given patient. The needle described has proved valuable at the clinic in performing satisfactory arteriography.

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